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MALFORMATIONS AND ANATOMICAL VARIATIONS SEEN IN THE MIDDLE EAR DURING THE OPERATION FOR MOBILIZATION OF THE STAPES.*

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INTRODUCTION.

The purpose of this thesis is to present some of the variations and abnormalities which may be seen in the middle ear during transaural tympanotomy. Mobilization of the stapes is the most frequently performed contemporary operation, using this exposure. The conditions here reported and discussed were observed in the course of 500 such procedures.

The importance of minutiae increases as the otologist's scope broadens, and his field of operation is seen with greater magnification. Numerous procedures[74,130,140,149,169,170] to rebuild, alter, or bypass the conductive mechanism of the middle ear have produced a pressing need for more information regarding the variations seen when the eardrum is lifted, and the complex mechanism of the middle ear is viewed. Microscopic vision and intense light have revealed structures which have stimulated otologists for decades. Now, the operator may engage in micro-orthopedics and plastic revisions of delicate proportions. Variations of as little as a fraction of one mm. or a few degrees have become more

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important than ever before. A slight distortion or the absence of a landmark can be a pitfall for the newly trained surgeon, and sometimes an experienced operator may find his way down an unmarked road into a disastrous result.

The report of variations found in the middle ear, or even a segment of it, from the outset is doomed to be incomplete. Exhaustive studies of only one small part,[10,14,17,16,18,23,11,13,15,9,35,12,20,19] such as the stapes, have occupied men over long periods of time. Study of variations of the ear is, however, important enough to justify the effort from a purely didactive biologic standpoint, but this justification is now reinforced by the currently evident clinical application; furthermore, thoughts regarding the development processes and theories of function may be stimulated in the minds of the readers.

In the past the discovery of middle ear anomalies unassociated with other known defects of closely related structures has been extremely rare.[5,6] Almost all of the reports in available literature have been of patients with atresia of the external ear with or without other defects of the entire hearing organ.[54,110,91,28,34,118,40,77,94,76,111,25,99,95,60,100,71,101,107,53,103,119,72,104,105,55,57,70,106,96,122,2,22,87,107,108,133,159,114,126,45,123,124,125,79,113,92,154,6,112,21,69,132,68] It is, however, my opinion that isolated malformations of the middle ear are not nearly so rare as has been previously thought; indeed, I am sure that many of these have been observed repeatedly although not yet reported. A large number of variations and abnormalities have been observed by me in 500 operations, so it seems reasonable to assume that these are not rare. The otologic surgeon, therefore, must constantly remember that a normal external canal may be approached and a normal tympanic membrane reflected only to unveil architectural and mechanical problems of several types.

SOURCES OF MATERIAL.

The reported variations and anomalies listed in this thesis were all found during the course of 500 transaural tympanotomies, most of which were done for the purpose of mobilizing the stapes. There will be no attempt to broaden the field of

this discussion to include those abnormalities of the middle ear which are so often found in association with other anomalies of the external or internal ear or other branchial derivatives.

These malformations, limited to the middle ear, are documented here by actual photographs taken at the time of operation. In order not to distort the color photographs by labels, sketches of anatomical structures of interest, are mounted beside each photograph, and the structures identified by appropriate pilot lines. The photographs were taken through the Zeiss operating microscope, using daylight Kodachrome film. The light and the objectives were fixed. The F-setting of the camera was 22. The pictures were taken in 1/50th of a second. The prints were enlarged in color directly onto a celluloid background.

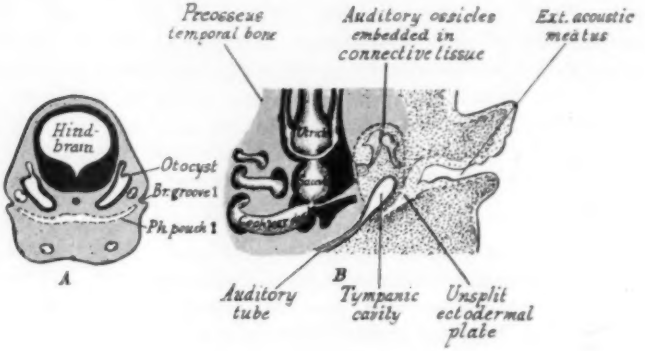
All of the literature available to me, both foreign and domestic, concerning embryology, physiology, anatomy, and malformations of the ear has been reviewed for the past 100 years. Local hospital and university libraries and national service organizations have aided in obtaining this literature. Photostats and microfilms have been secured of rare and nontransferable articles from such services as the National Library of Medicine, the Armed Forces Medical Library, etc.

ORGANIZATION OF MATERIAL PRESENTED.

A presentation of the visual experience of these variations and malformations requires some discussion as to the significance of what is seen. First, a discussion of fundamental embryology of the area will be given. Next, consideration of the individual variations seen in the following structures of the middle ear will be given: *1.* Muscles and Tendons; *2.* Bones and Joints; *3.* Nerves; *4.* Vessels; and *5.* The Round Window. Photographs and drawings of ears, demonstrating some of the variations and abnormalities, will then be presented in the same order. A brief explanation regarding the purpose of presenting each photograph will be given. Comments that seem appropriate will be offered regarding surgical significance, etiology, embryology, and case history.

Finally, general discussion under the headings of, first, etiology, and second, diagnosis, of malformations of the middle ear will be presented.

Embryology—(see Fig. 1). Drawing from diagram in Arey, "Developmental Anatomy," W. B. Saunders Co., 1940.



Progressive association of the primordia of the external, middle and internal ears, illustrated by partly schematic sections. *A*, At six weeks; *B*, at three months.

Fig. 1.

THE EXTERNAL EAR.

The external ear is formed by a modification of the tissues of the median portion of the first branchial groove and its neighboring first branchial arch (mandibular) above, and the second branchial arch (hyoid) below. The auricle is formed by several knoblike structures that appear on the first two arches about the sixth fetal week. These are gradually molded together to form the adult auricle.

The external auditory canal in reality represents the first branchial groove. The membranes (ectodermal) of this groove are for a time (fourth to the fifth embryonic week) in contact with the internal (entodermal) tissues of the first pharyngeal pouch. This contact is soon lost, however, by the

expansion of the head, which tends to separate the primitive external ear from the middle ear. Mesodermal tissue separates the two epithelial layers during the ossicular development. Near the end of the second month of fetal life, the ectoderm thickens at the bottom of the groove and grows toward the middle ear. This solid meatal plate remains closed until the seventh month, at which time it splits and forms a canal. The innermost portion of this plate becomes the superficial layer of the tympanic membrane.

THE MIDDLE EAR.

The tympanic cavity and the auditory (Eustachian) tube actually represents a drawn-out first pharyngeal pouch. The second pharyngeal pouch may also be merged with it.[53] The surrounding tissue of the first and second branchial arches constitutes the primordia of most of the other tympanic structures, such as ossicles, muscles, tendons, and connective tissue.

The first pharyngeal entodermal pouch appears late in the third embryonic week (3 mm. stage) and expands rapidly. For a time it is in contact with the ectodermal membrane of the external first branchial groove (primitive external canal). This long open tube undergoes a bottleneck constriction near the end of the second month. The constricted portion lengthens and becomes the auditory (Eustachian) tube, whereas the blind outer end of the pouch expands to form the tympanic cavity. At eight weeks (23 mm. stage), the tip of the flat tympanic cavity lies alongside the ectodermal tissue of the first branchial groove, which is the future external canal. These two cavities are separated by mesenchyme which later becomes the manubrium of the malleus and the middle fibrous layer of the tympanic membrane. Above, medial and posterior to the area of the future expansion of the tympanic cavity, the other ossicular and muscular elements are being developed from the mesenchymal tissue. As these structures progress to maturity, the unused surrounding mesenchyme disintegrates and the entodermal-lined tympanic cavity expands. This expansion is in pace with the concomitant development of the cartilaginous otic capsule and the external

ear canal. As the tympanum expands the lining epithelium surrounds the ossicles and muscles. By the 30th week (270 mm. stage), the cavity formation of the tympanum is complete, but another month is required before the epitympanum is near completion. At term most of the expansion is finished and the cavities are almost mature.

OSSICULAR AND MUSCULAR DEVELOPMENT OF THE MIDDLE EAR.

This thesis is so intimately concerned with the ossicular and muscular structures of the middle ear that a more detailed discussion will be made regarding their known developmental processes. A brief chronologic story of embryologic development has been correlated from several authoritative sources.

The stapes is the first ossicle to make its appearance. When the embryo is four-and-a-half weeks old (7 mm. stage) there is a concentration of mesenchymal cells at the cranial end of the second branchial arch, also referred to as the hyoid, or second visceral bar. This is situated lateral to the mesenchyme of the primitive otic capsule, and is separated from it. It is still connected with its branchial arch (Reichert's cartilage, or hyoid bar). When the embryo is six weeks old (12 mm. stage), the stapes is represented by a dense mesenchymal ring penetrated by a large stapedial artery.

At the end of six-and-a-half weeks (14 mm. stage), the stapes is continuous with its branchial arch (Reichert's cartilage) only at the lateral portion, which will become the future head of the ossicle. It is at this age that Meckel's cartilage of the first branchial, or visceral, arch is seen (also called the mandibular arch). This will rapidly differentiate into the malleus, the incus and the tensor tympani muscle.

When the embryo is seven weeks old (17 mm. stage), the stapes is annular in shape like a doughnut. It has moved into close apposition with the lateral wall of the otic capsule. The basal portion of the stapes abuts the otic capsule and produces a concavity in it. The stapes is still connected with its visceral branchial arch (hyoid), and it is from this remaining connection that the stapedius muscle and tendon

will be formed. At this age the malleus and incus are recognizable, and the incudomalleolar joint begins to separate the two ossicles. The incus is beginning to approach, but not touch, the head of the stapes for the formation of the incudostapedial joint. Precartilage may be seen in the tissue of the ossicles and the otic capsule.

In the eight-week-old embryo (23 mm. stage), the stapes is cartilaginous, and its base is pressed hard against the otic capsule. The remaining visceral bar connection becomes a strong mesenchymal band. This band attaches to the lateral mass of the otic capsule, thus connecting it with the stapes head. This will become the stapedius muscle. A very small stapedial artery is still present. The malleus is still connected to Meckel's cartilage, and the incudomalleolar joint has developed an articulating surface. The joining of the long process of the incus with the stapes head has occurred.

The embryo, at the age of nine weeks (30 mm. stage), has true cartilage forming the stapes and the otic capsule, except in the vestibular region of the capsule against which the stapes rests. This area is called the lamina stapedialis. This portion of the capsule actually has two cellular zones. The more superficial is mesenchyme which lies against the perichondrium of the base of the stapes. The deeper layer is fibroblastic and blends with the mesenchyme supporting the epithelial labyrinth. Actually this lamina stapedialis fuses with and becomes a structural part of the footplate of the stapes; therefore, it may be said that a part of the otic capsular wall contributes to the formation of the stapes.

Between the 12th and the 14th fetal week (50 and 75 mm. stage), the crura of the stapes become bowed, and the union of the incus and stapes differentiates into an articulating joint. The base of the stapes has now become a part of the lamina stapedialis and blends with the surrounding capsular tissue. This blending zone gradually undergoes further change into the annular ligament. Actually, an orifice as such never exists, since the stapes is histologically continuous with the marginal cartilaginous vestibular window. The annular ligament is first composed of chondrogenic tissue but progressively changes to a fibrous tissue.

When the embryo is 16 weeks old (112 mm. stage), the ossicles are still cartilaginous. Ossification then appears first in the incus along the anterior aspect of the long crus.

By the 17th week of embryonic life (125 mm. stage), bone makes its initial appearance in the body of the malleus. It is also at this time that ossification begins in the otic capsule.

At about the 19th week of embryonic life (150 mm. stage), ossification begins in the stapes by the appearance of a single center on the obturator surface of the base. From this center, ossification spreads over the base and along each crus.

At about the 28th week of embryonic life (225 mm. stage), ossification has occurred throughout most of the ossicles. The usual evacuation of the stapes is beginning to occur along the obturator margins. This progresses until the crura are reconstructed into channeled troughs, the head is hollowed to form a cylinder, and the base becomes a thinned bilaminar plate. This exceptional differentiation is found only in the stapes. The malleus and incus, like all of the other bones of the body, progressively become thicker through the development of endochondral bone whereas the stapes becomes thinner, loses bulk and disposes of its marrow tissue. Cartilage remains throughout life on all articulating surfaces of the ossicles. Cartilage still covers, and will persist, on the vestibular surface of the base of the stapes. Cartilage still remains and will persist throughout life in the rim lining of the oval window.

By about the 35th week of embryonic life (345 mm. stage), the stapes has assumed adult form, as have also the malleus and incus. Mucous membrane has lined the spaces of the middle ear and is draped over the tympanic surfaces of the ossicles.

At term (367 mm.) the adult form of the ossicles is present and their relationships are established. The ossicular muscles are fully developed and the mechanism of sound transmission is established for life's sojourn on earth.

THE INNER EAR.

The inner ear is produced from epithelium derived from ectoderm. Very early in embryonic life (three to four weeks, or the 2 to 4 mm. stage) there develops an ectodermal thickening called the auditory placode on the lateral surface of the head. This area invaginates to form a deep pit, the orifice of which soon starts constricting and eventually closes. This forms a closed sac of trapped ectodermal cells, called the otocyst. This becomes separated from the surface of the head and is completely surrounded by mesenchyme. At the fifth week (8 mm. stage), the otocyst begins differentiation into the vestibular and cochlear portions. The cochlea begins to coil at the seventh week (17 mm. stage), and the vestibule has developed the primitive utricle, saccule, and semicircular canals. The surrounding mesenchyme becomes the otic cartilaginous capsule which begins ossification at about the 17th week of fetal life (125 mm. stage). The elaborate embryologic differentiation of this highly specialized structure will not be traced further, as it is not needed for the purpose of this thesis. The differentiation has been completed, and the organ is mature at birth.

VARIATIONS—MUSCLES AND TENDONS.

"Malformations of the muscles of the middle ear, especially their partial or complete lack, have been observed only when associated with other severe malformations."[93] Again, this condition is not so rare as has been previously considered. During the course of 500 operations, I have found five patients with a total absence of the stapedius muscle, tendon and pyramidal eminence (see Figs. 16, 5, 6, 7, 8). In one patient (see Fig. 13), the muscle band was not much more than a condensation of soft tissue.

The significance of abnormalities or absence of the stapedius muscle has been the object of highly specialized scientific investigation.[166,117,90,163] Apparently the stapedius muscle has the ability to reduce or dampen sound transmission en route from the tympanic membrane to the cochlea. Some believe a reflex action may enhance certain sounds as well as

reduce certain intensities.[163] Others believe that it is not a muscle of "attention," but a protective adjustor.[90] The extent of the true value of the stapedius muscle is certainly not known, but now we can at least be sure that it is not absolutely necessary for serviceable hearing. This is proved by an example of the return of hearing in a patient with ankylosis of the stapes and concomitant absence of the stapedius muscle (see Fig. 5). After mobilization of the stapes, this patient's hearing returned to a good serviceable level. This patient seems to have had no difficulty with tolerance to intense sounds, pitched either high or low and, therefore, does not seem to miss the "protective" mechanism.

A case has been previously reported of ossification of the stapedius tendon producing stapes ankylosis.[142] This has not been observed in this series, but embryologically one might expect this change rather frequently. The stapedius head is joined to the hyoid bar rather late, and this tissue eventually attaches to the otic capsule and becomes the stapedius muscle, tendon and pyramidal eminence, with bone being formed on both ends and around the muscle belly. All these structures are derived from the same branchial tissue and one could expect, occasionally, a differential change from muscle to bone.

Supernumerary muscles have been reported in the bony facial canal.[4,30] This muscle is without insertion and apparently is purposeless. Other small muscle fibers have been seen in the substance of the temporal bone, but none has been observed in the middle ear, and none was found in this series.

Variations in the direction from the insertion of the stapedius tendon into the stapes anteriorly to its origin in the pyramidal eminence posteriorly are frequently found. They are of interest only in that they might hide a certain view of the posterior crus and footplate of the stapes by such a location. In the majority, the tendon extends directly posteriorly and approximately parallel to the posterior crus (see Fig. 2). In a few instances, it will be directed inferiorly and posteriorly (see Fig. 3), and very rarely it will be directed superiorly and posteriorly. The visualization of the pyra-

midal eminence during the operation for mobilization of the stapes without bone removal from the external canal wall is quite variable. Although frequently it may be seen in its entirety (see Fig. 2), usually the chorda tympani nerve must be dislocated, and some canal wall bone must be removed before it can be completely inspected (see Fig. 27).

Variations in the length of the stapedius tendon are great. Some are extremely long (see Fig. 31). Some are almost too short to see (see Fig. 4). The unusual projection of the stapedius pyramidal eminence and the extremely short tendon seen in this photograph are extremely rare. The manipulation of the stapes in an operation for mobilization of the stapes can be interfered with by this close proximity. Visualization of the movement of the stapedius tendon is sometimes used to identify certain slight motions of the head of the stapes during mobilization.[130,140] The size, location, and exposure variations are important in this light.

The attachment of the stapedius tendon is to the neck of the stapes in about three-fourths of ears. Occasionally, it is attached to the head or to the posterior crus (see Figs. 17, 2, 3). Usually the tendon is attached to a spine on the stapes.[24] One writer believed that the spine was the result of a traction process, whereas another felt that the spine was perhaps due to ossification of a certain portion of a tendon.[142] It is my belief that neither is true, since I have observed several cases in which a rather long spine was projecting in a different direction from the line of the tendon, and the tendon was attached to its distal end. If this spine had resulted from either a traction process or a tendon ossification it would have been in line with the tendon.

Discussion of middle ear muscles in this thesis will not include the tensor tympani muscle because it is not usually observed in trans-aural tympanotomy.

BONES AND JOINTS.

Variations of the ossicles in an otherwise normal auditory organ are rare.[23,26] The stapes is probably the most frequently affected ossicle,[93] but the vast majority of reports in the

literature associate ossicular deformities with other severe head and branchial arch deformities. In reviewing the literature, many of those reported were found in animals.[26,24]

THE STAPES.

"In spite of the importance of the stapes, no single element in human anatomy has suffered more chronic neglect. This is a strange instance of disregard, since the stapes has great clinical importance in otosclerosis. It passes through developmental stages of extreme interest, and is grossly the most captivating bone in the human skeleton. For all of its unique fascination as the smallest essential bone and as the most exquisitely fashioned unit, this ossicle has not only been forgotten by investigators in anatomy and otology, but the descriptions of it in standard textbooks of anatomy have become stylized and debased through the years. They are inaccurate and fragmentary, commonly perpetrating century-old errors. Illustrations are either minute or diagrammatic."[24] Bearing this quotation in mind, an objective description of the unusual anatomy of this bone will be presented, with the discussion of the variations mentioned in the literature and encountered by me in this series.

THE HEAD OF THE STAPES.

The head of the stapes is normally broad and shallow,[44] and its surface is elliptical and concave for articulating purposes. This surface is usually in a near sagittal plane, thus permitting the use of pressure through the incus on the stapes without causing one part to slide on the other, thus producing possible joint separation (see Fig. 20). Occasionally, however, the joint will be tilted forward, permitting anterior dislocation if transincus pressure is applied; also occasionally the joint face of the stapes will be facing at a downward angle as though the long process of the incus had overgrown itself, and the joint formation was an afterthought (see Fig. 6). Another variation is for the head of the stapes to be tilted superiorly, thus making the articulating surface face more in an upward plane (see Fig. 14). This would make one feel that the long process of the incus and the

stapes had almost fallen short of union, and the stapes had turned its head up for a slight distance advantage. Indeed, the union of these two bones, which are embryologically derivatives of separate branchial arches, would make one wonder that much greater variation does not occur. The usual incudostapedial joint is found with the lenticular process of the incus placed slightly more anterior than the head of the stapes. This allows the posterior rim of the head of the stapes to be clearly seen. Frequently the lenticular process is placed squarely on the head and occasionally more posterior than the head of the stapes (see Fig. 2).

The head of the stapes may be extremely hollowed-out or solid, and may be as a rounded knob on a pedestal, or merely a flattened surface on the top of an arch formed by the crura. The normal and the usual is somewhere between these extremes. Bone erosion may produce a variety of knobs, spines, and holes in the head or the neck of the stapes.

The neck of the stapes may be thick or thin, solid or hollow, strong or weak.[24] Its length does not always determine its strength. A long slender neck may well stand a great amount of pressure which might be exerted on it during operative maneuvers.[130,140]

The head of the stapes is usually tilted slightly forward in relationship to the crural arch; however, it frequently meets the arch in a true perpendicular manner, and is occasionally tilted slightly posterior.

The clinical significance of these variations is obvious.[3] If the articulating surface is markedly tilted, efforts to perform a mobilization of the stapes by pressure exerted through the incus will frequently cause distortion and lysis of the joint, whereas recognition that there is a moderate tilt in one of the above directions will allow the surgeon to apply force in a direction that will allow for the tilt. It has been my experience that the two most dangerous positions in this respect are a tilting of the head of the stapes downward or forward. In some instances, mobilization through the incus is impractical. Picks in the head of the stapes are sometimes used to mobilize a fixed footplate. If the incus is placed unusually

far posterior it may be impossible to engage the pick in the head of the stapes without joint dislocation. Often the head and neck of the stapes have been rendered a hollow shell by bony absorption from the obturator surface. The outside appearance may be that of a heavy solid structure, but the application of even a fine pick may break through the shell and cause the head to crumble partially.[74]

One maneuver for mobilization of a fixed stapes depends a great deal on the structure of the head and neck as well as the crura. The pressure in this maneuver is against one area of the anterior portion of the neck or head, and the line of force is in a posterior direction.[130] The head of the stapes is usually tilted slightly forward, and this is an advantage in the application of force in this direction.

THE CRURA.

The stapes crura are long arched structures which fuse into one at the neck of the stapes, and are widely divergent as they meet the footplate. They are hollowed on the inside, or obturator surface, making them an inverted rounded trough, the edges of which become more pronounced as they curve to meet the footplate. This trough is usually deeper at the base of the posterior crus than at the base of the anterior crus. This hollowing out is the result of bony absorption and can produce a large number of variations. The crura may be extremely hollowed out to a very weak thin shell; on the other hand, there may be no visible evidence of erosion so that the crura are left very solid and large. This is an infantile type of crura. Bony projections, spurs and eroded-looking pits may frequently be seen, especially on the obturator surface.

The anterior crus is usually straighter and shorter than the posterior. The anterior crus averages 3.62 mm. from the head to the base. The posterior crus averages 3.73 mm. from the head to the base. The anterior crus is the more slender of the two. This description is considered to be almost an invariable one by most authors. There seems to be only one who has disagreed with this description.[75] At least one can say that

it would be a very exceptional case in which the posterior crus would be straighter and more slender and shorter than the anterior crus (see Fig. 5). Actually rare specimens occur in which the anterior crus is longer than the posterior and fairly commonly the crura are equal in length and about alike in curvature (see Fig. 31).

Very little reference is made in past literature to a finding which I have frequently encountered. These cases are those of crural bowing, either inferiorly or superiorly. They may seem to wrap around the curve of the promontory or be curved around the bony facial canal[6] (see Fig. 6). Bowing of the stapes crura is probably more frequently seen in a downward direction. The intimate bowing around the bony facial canal is extremely rare (see Fig. 14). Bowing of the crura must be considered if stress is to be exerted in any direction during an operation.

Bony projections may be seen frequently on the surface of the posterior crus. A knee-like area may frequently be seen about one-third of the distance down from the head toward the footplate on the posterior crus.[24] There is frequently a bony spine on either the head, neck, or high on the posterior crus for the insertion of the stapedius tendon. Rarely one may see a heavy bony rod extending from the promontory or the facial canal to join the posterior crus, thus causing fixation to the labyrinthine capsule[1,52] (see Fig. 18). I have observed that it is common to see bony bridges which project from the promontory and are very near one of the crura. These are seldom fused with it (see Fig. 17).

Severe variations of the crura are very rarely seen.[23,24] They have been reported only with severe deformities of the skull. Most of the severe crural deformities reported in literature were seen in animals.[26,24] Isolated human anomalies are extremely rare.[26] I believe these malformations are far more common than is realized. Fusion of the stapes crura into a double-barreled stapes was observed one time in this series, and has been observed previously.[24] This is perhaps due to variation in the primitive stapedial artery or abnormal ossification of the inner crural membrane, or due to a variation

in bony absorption. In Marmozets, the crura of the stapes are often found fused for a short distance below the head.[44] In Montremata, which is a true mammal of a low skeletal form, the stapes is normally absolutely columelliform (unicurate stapes).[44]

Figure 15 represents a stapes with only one crus. This is the posterior crus with the tympanic side of the base being visible in the photograph. The incudostapedial joint has been disarticulated in this ear, and the head of the stapes rotated posteriorly so that the basilar obturator surface of the posterior crus could be seen as it joins the footplate area. The crus was fairly well developed, but there was no footplate. There is a total absence of the anterior crus, thus making this a completely unicurate stapes. This finding was in a patient with unilateral deafness.

Complete bony union of the crura may be seen, due to massive otosclerotic mounds filling the obturator space (see Fig. 19). In the case of a deep niche, the pathologic bone formation may fill in the niche for the stapes and obscure the landmarks below the stapes neck and upper arch. This amount of proliferation is rare. In certain anomalies, the crura may be extremely small and fragile (see Fig. 14); in other conditions the stapes may be extremely short and bulky (see Fig. 4). In the short bulky stapes the obturator foramen may be very small and the stapes may be almost like a rounded blob of bone (see Fig. 4). This type of stapes, obviously, has not developed even to the stage usually seen in later uterine life. In late uterine life the stapes is even larger than the adult form and must undergo reduction in size by bone absorption, whereas in this patient the stapes is even smaller than usual, but it has much thickness and, therefore, represents a very early embryologic stage.

The crura may join the footplate at a variety of different angles. Their junction with the footplate may be at right angles or the crura may be tilted inferiorly or superiorly to the plane of the footplate. Usually, the footplate of the stapes is in the vestibular window at an angle slightly off the sagittal plane of the body. This may vary to an almost horizontal

plane as seen in Fig. 14. In this patient there was a severe congenital malformation of the labyrinthine wall, making the area of the stapedius laminalis almost look down on its tympanic aspect. This is because the window area is situated under and medial to the facial canal. At the other extreme is the footplate that is flat or looking slightly upward, as seen in Fig. 31.

The bony overhang of the facial nerve above or the promontory below determines the direction and width of the niche in which the stapes must fit. The crura are usually attached to the footplate at a slightly superior angulation *in situ*.[23] This is an unusual statement since in a large majority of cases the footplate is seen mostly above the crura and behind the long process of the incus and below the overhang of the bony facial canal; thus one would expect the angle of insertion of the crura to be more inferior. This is due to the fact that the vestibular window is at an angle of inclination even greater than the angle of the crura; therefore, the crura are attached to the footplate usually at an angle of less than 90 degrees from the superior edge of the footplate to the crura.

The trough margins of the obturator surface of the crura extend in varying degrees out on the marginal edges of the footplate. These lips may run completely around the footplate to produce a rim or tray edge as in the infantile stapes, or the trough margins may be hardly perceptible on the crura and absent on the footplate. In my experience, it is unusual to see this crural trough edge extending onto the margin of the footplate. Rarely a small bony ridge is seen on the tympanic surface of the footplate extending between the crura. This is called the crista stapedius and is very inconstant. It is reported that at times the ridge may be replaced by a crest.[23]

THE FOOTPLATE OF THE STAPES.

The footplate is usually rounded in an arch on its superior edge, curved with a slight point anteriorly, almost straight on its inferior edge, and gently curved on the posterior edge.[24] The footplate may frequently be extremely narrow and long, being wedged into a narrow niche between the facial ridge

and the promontory. It may frequently be generously wide with much of its area being superior to the crural insertion (see Fig. 10). Rarely the inferior margin is seen in its completeness (see Fig. 31). There may be a slight extension of the footplate, either anterior to the anterior crus or posterior to the posterior crus. In one patient, I observed a marked extension posterior to the posterior crus of about 1 mm. This shelf was proven to be present on mobilization by visualizing its free margin.

The average length of the base of the stapes is 2.99 mm. with variations of from 2.64 mm. to 3.36 mm. The average width is 1.4 mm. with variations of from 1.08 mm. to 1.66 mm.[24]

The annular ligament connects the circumference of the stapes to the rim of the oval window, and is very frequently the site of otosclerotic bony invasion, especially in its anterior area.[168,31] Due to the embryologic development of the annular ligament, which is derived from the same tissue that later becomes the bony labyrinth on one side and the footplate of the stapes on the other,[24] it is reasonable to assume that free cartilage or actual cartilaginous nests may devleop and remain within its substance.[93,28] These nests might become osteogenic and produce bony deposits capable of causing footplate ankylosis. Thin bridges of cartilage in the annular ligament presumably could become ossified in the middle decades of life much as cartilage in other regions of the body undergoes such change.

The thickness of the footplate is the most variable of the areas considered. An extremely thin, almost transparent, footplate is frequently seen. On the other hand, a thick, strong, opaque footplate is common.[131] These extremes seem to be normal variations. Otosclerotic infiltration may involve a small rim area, or may when advanced, change a portion of the footplate into heavy otosclerotic bone and leave the remainder quite thin and normal. The anterior areas are the most commonly infiltrated. Smooth infiltration of the entire stapes and annular ligament so as to obliterate all fenestral and footplate identification may be seen (see Fig. 22). Oc-

casionally, the otosclerotic bone is seen in mounds occupying isolated areas, or to a massive degree as mountains filling in the entire niche and covering large portions of the crura (see Fig. 19).

THE LONG PROCESS OF THE INCUS.

Only the long process of the incus is visible during the ordinary operation for mobilization of the stapes. It projects from under the superior canal rim, usually as a long, slender, slightly convex process which bows slightly anteriorly and gradually becomes more medial toward its tip. The end of the tip has a structure designed for articulation with the head of the stapes. This structure is a small mass of bone which extends medially from the main shaft and bears a flat elliptical disc with a joint surface of cartilage. A variety of names have been given to this portion of the long process of the incus, such as the lenticular process, the *os orbiculare* or the Sylvian apophysis.[44] Some authorities have considered this a separate bone, but others have held that it is only an epiphysis of the incus.[44]

The lenticular process may assume a variety of shapes which are important in present-day surgical procedures in the ear. A structure much like the head of the stapes may project medially from the long axis of the process. It may rest upon a small but distinct neck and may deviate anteriorly or posteriorly, superiorly or inferiorly, in order to articulate with the stapes. The joint surface may be in any of the planes described under the head of the stapes variations, and are important because of the reasons listed there. Bony strength of this head and neck are important, due to the possibility of fractures during various operative maneuvers.

Often the long process of the incus does not have an articulating head and neck, and there is found only a flat articulating surface on the medial side of the long process of the incus.

The lateral side of the long process of the incus may have a variety of bony projections. A spine-like structure may be seen on the lateral surface at the very end of the process (see

Fig. 24). This usually points slightly anteriorly, and gives the appearance of the bow of a ship. Frequently, the opposite of this may be seen, in which the process rapidly tapers off at the end as it meets the stapes, giving a rounded-off appearance (see Fig. 23). The application of various instruments for transincus pressure on the stapes will depend on the direction, topography and strength of this area. Slipping-off or fracturing can result from lack of consideration of these variations.

Erosion or necrosis of the lenticular process by disease will be discussed under etiology and with the presentation of the case in Fig. 21. I have seen complete erosion of the lenticular process of the incus three times in this series in the presence of a normal eardrum and canal. I am sure that it will be seen much more often in the future than in the past. This necrosis, if complete, produces a gap in the ossicular chain and results in an air conduction hearing loss of about 60 db.

A total separation of the incudostapedial joint without necrosis is reported in this series. Discussion and descriptions are treated elsewhere. This is the second patient reported in the literature available to me.

In this series, variation in direction of the long process of the incus as it projects downward is at least 45 degrees.

Bony fusion of the lenticular process of the incus with the head of the stapes and the handle of the malleus is a rare anomaly, and is presented in this series (see Fig. 8). As far as I am able to ascertain, this is the first case of such abnormality reported in the literature. This was a huge bony mass which involved all three ossicles. Such an anomaly requires a triple arthrolysis before a mobile sound-transmitting apparatus can be obtained.

A much simpler fusion at the incudostapedial joint may occur unassociated with any other abnormality. Numerous reports have been made of simple joint fusion of the incudomalleolar joint, but I have found no reports of incudostapedial joint fusion. The reason, I believe, is not because it is more

rare, but because this area has not come under observation in the past as often as the epitympanum. I have encountered one such frozen joint between the incus and stapes in this series.

A review of the literature on the histopathology of ossicular articulation and particular discussion of the findings of foreign authors has been done.[46] It was found that there were four distinct layers, almost constantly found in all ossicular articulations. They are from the bone outward: *1*. The bony layer; *2*. The layer of calcified cartilage blending with bone; *3*. A layer of hyaline cartilage; and *4*. The terminal or meniscoid articulating surface. A very interesting finding has been the lack of disease in the joints, even in a large number of patients who had middle ear suppurative disease. It has been considered, however, that actual ankylosis of the incudomalleolar joint is usually the result of inflammation.

NERVES.

Only the facial nerve and its chorda tympani branch needs to be considered in this thesis. Marked variations of the position of the facial nerve in its horizontal course across the middle ear are extremely rare.[93] Minor variations are common, and are of great importance in the region of the oval window where present-day instrumentation is common. There usually is a definite bulge of the bony facial canal just lateral to and above the footplate of the stapes. This bulge is usually not sufficient to hide the superior margin of the footplate. Marked inferior and lateral bulging occasionally hides a part of the footplate and crowds the crura against the promontory below. Obviously, this will hinder instrumentation, and may predispose to nerve injury if forceful entry to the footplate area is made. The facial nerve in its bony covering may present only a very small bulge in the labyrinthine capsule wall, which will represent a nerve of small diameter, or one that is deeply placed in the bone (see Fig. 18). The opposite extreme is the huge nerve and bony canal that can be seen in Fig. 16. This is an unusually large fallopian ridge.

A bony dehiscence in the facial canal wall is of great importance. These are not uncommon in my experience, and invite injury to the facial nerve by either mechanical injury or inflammatory involvement.[93] Usually the dehiscences are located just above the oval window[93] and are covered with a layer of connective tissue and mucous membrane.[144] They are usually oval in shape and the soft tissue may bulge out of the opening slightly.[50] "This is explained by the fact that the facial nerve originally lies in a sulcus of the labyrinthine capsule, which is closed to form a canal by the formation of a bony cover or a connective tissue base in late embryonic life and the first year of the post-embryonic life."[50] Defects of ossification may occur, especially in connection with a hyperplastic condition of the mucous membrane of the middle ear.

The chorda tympani may be very large or extremely small in diameter. It usually enters the bony external ear canal (Iter chordae posticus) on its edge just lateral to the stapedius tendon. Occasionally, the nerve enters its bony canal 1 or 2 mm. external or lateral to the external canal edge. This was observed in three ears of this series. There may be a slight variation in the location of bony entrance on the rim of the canal wall. Using the right ear as an example and considering the top of the head to be the 12 o'clock position, the chorda tympani may enter its bony canal between 9 o'clock and 10 o'clock. It is usually at a position just superior to the end of the fibrous annulus of the tympanic membrane. As the nerve continues superiorly along the canal rim it seems to function also for a short distance as an extension of the fibrous annulus of the tympanic membrane. A varying amount of bone covering is found over the nerve along the rim.

No extensive hypoplasia or absence of the facial nerve or the chorda tympani was observed in this series, and I have found no reports of malformation involving the nerves alone in the literature available to me. A marked hyperplasia of the facial nerve, or its bony covering or both, is reported here in Fig. 14. Malformations of the facial nerve may be found in conjunction with other severe anomalies.[50,4] Usually

the anomalies reported have been of the descending portion of the facial nerve and not the horizontal portions.

VESSELS.

Vascular anomalies of the middle ear are extremely rare but must be constantly kept in mind. Among the anomalies, the so-called persistent stapedial artery is one of the most commonly known.[93,30] This artery is a persistence of a primitive vessel which is a branch of the internal carotid artery. It perforates the floor of the tympanic cavity and progresses over the promontory, passes between the crura of the stapes and enters the facial canal. It later enters the cranial cavity and divides into branches to the dura.[93,158] I have not encountered a persistent stapedial artery in this series. Although it has been commonly reported that this could be the cause of some types of tinnitus, I could find no documented reports of human cases in the literature; however, there have been many references to this anomaly in animals.[158]

Another common anomalous artery which should be mentioned, originates from the superficial petrosal branch of the middle meningeal artery. This vessel may accompany the facial nerve and may be found in the mucous membrane along the posterior wall of the niche of the oval window, passing through the crura and branching into a plexus on the promontory. I have not observed this anomaly.

Branches of the vestibular artery may be in close proximity to the labyrinthine surface of the footplate of the stapes. This artery is a branch of the internal auditory artery. Profuse bleeding from the vestibule may occur by accidental tearing of this vessel during mobilization of the footplate. If the vessel were so closely bound down to the footplate as to cause tearing of it during manipulation it would be considered to be in a very rare anatomical position. The cause of profuse bleeding in the example to follow is not known, but it is assumed to be due to the above anatomical variation. During the mobilization of a stapes footplate in one patient of this series, the following sequence of events occurred: Instrumentation was proceeding via the head of the stapes

when suddenly a profuse hemorrhage occurred from the vestibular window. This occurred as the footplate was seen to become slightly mobile. This hemorrhage was definitely a pulsating type of bleeding, and was so rapid that within a matter of seconds the entire middle ear, the ear canal, and the ear speculum were filled with blood; it was so rapid that the suction could not handle its volume. This bleeding gradually subsided over a period of four to five minutes and eventually stopped entirely, never to recur. Although mobilization attempts were discontinued, and consequently there was little postoperative hearing improvement, the profuse hemorrhage apparently did not damage the labyrinth, since hearing deterioration has not followed.

Two other vascular variations should be mentioned although neither was seen in this series. It has been reported that, rarely, there is an absence or a dehiscence of the floor of the middle ear allowing the jugular bulb to herniate upward into the middle ear; also, vascular tumors, such as glomus jugulare, may arise in this area and should be remembered in diagnostic differentiation.

THE ROUND WINDOW.

Variations in the size, location and shape of the round window are interesting and sometimes clinically significant. Otosclerosis can cause complete occlusion of the round window.[89,142] It has been found that otosclerotic occlusion of the round window causes hearing loss comparable to fixation of the stapes.[161]

In this series I have encountered no cases of complete closure of the round window but have seen several cases of extremely small windows. Identification of otosclerotic foci at the periphery of the round window has not been possible under 16-power magnification, and these small windows observed are considered to be developmental. The size of the window seemed to have little to do with the clinical restoration of hearing in this series; however, it must be pointed out that no total closures were observed. Very large windows were occasionally seen, and a variety of membranes were

PRESENTATION OF PHOTOGRAPHS AND DRAWINGS AND DISCUSSION OF ANOMALIES AND VARIATIONS OF THE MIDDLE EAR

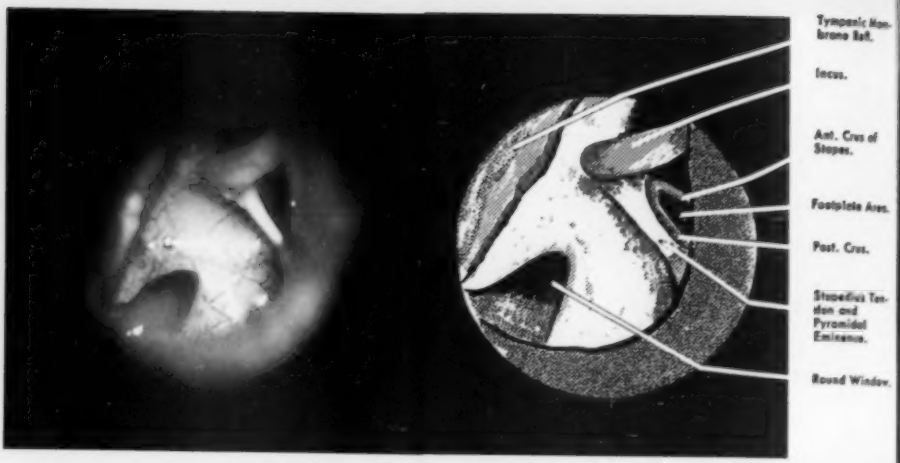


Figure 2.

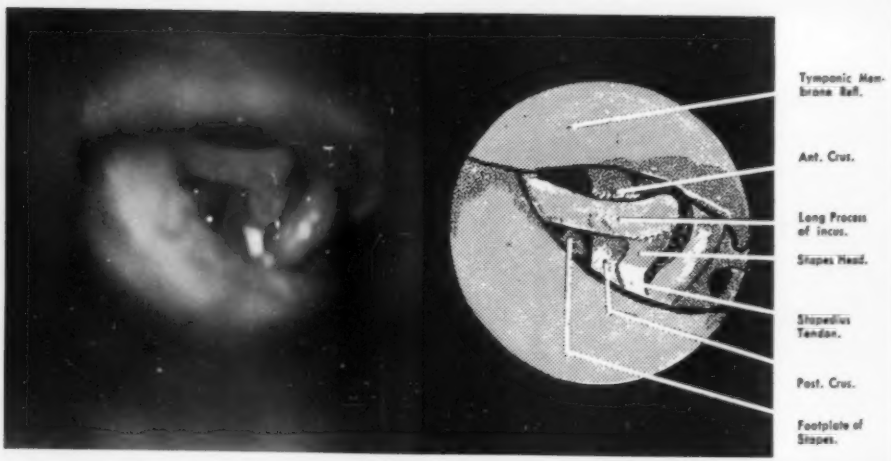


Figure 3.

Figure 2.

Photograph of a left tympanic cavity presented for the purpose of demonstrating variations of the stapedius muscle and its tendon.

This stapedius tendon is seen in beautiful contrast. Occasionally one sees the entire stapedius tendon and the pyramidal eminence without disturbing the chorda tympani nerve or the bony external canal. This tendon is of average length and is inserted directly into the head of the stapes. This tendon is parallel with the posterior crus, in its usual position.

Observe the excessive vascularity of the mucous membrane.

Figure 3.

Photograph of a right tympanic cavity displayed for the purpose of showing a variation in the tendon of the stapedius muscle.

The stapes is unusually large, as is the long process of the incus, yet the stapedius tendon is relatively small. The tendon is inserted directly into the posterior crus of the stapes. Although this variation is not uncommon, it is the most unusual point of insertion. The neck and the head are the most frequent sites for stapedial tendon insertion.

Usually, the tendon of the stapedius muscle is parallel to the posterior crus of the stapes, but in this ear it is deflected at almost a 45 degree angle inferiorly. Even more uncommon than this position is that of a deviation of the tendon in a superior direction.

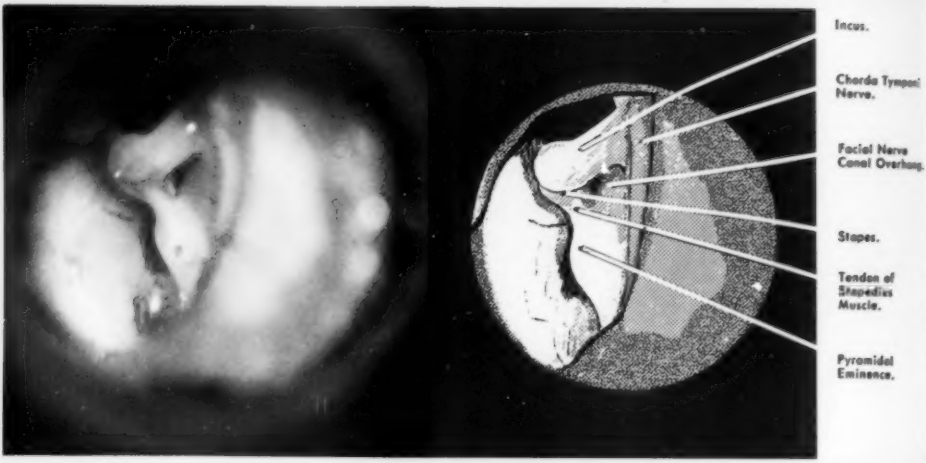


Figure 4.

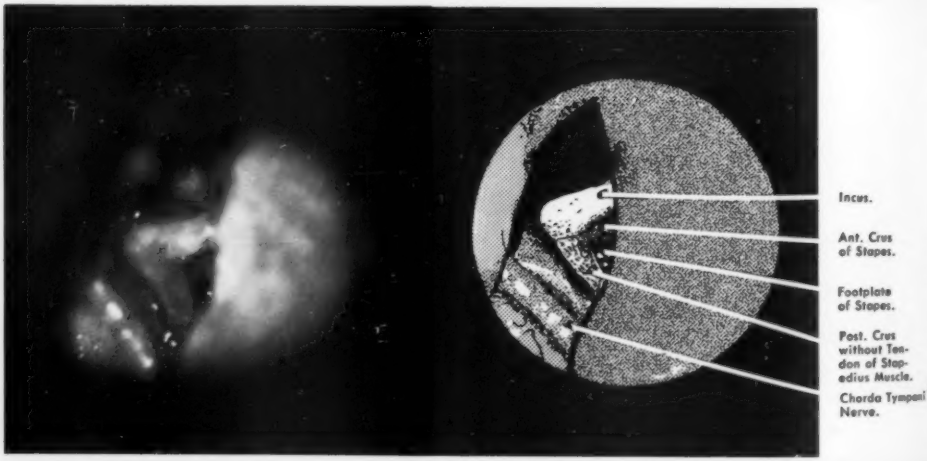


Figure 5.

Figure 4.

Photograph of a left tympanic cavity displayed for the purpose of showing bony and muscular variations.

The long process of the incus describes an S-curve and articulates squarely with the head of the stapes.

This stapes is extremely small, yet the crura are very thick. An obturator foramen, if present, is so small that a right angle pick could not palpate it; it could not be seen. This stapes seems to be a rounded mass of bone in a very immature state. Late in fetal life, the immature stapes is much larger than the mature stapes. This apparent paradox is probably explained by the cessation of development in this patient somewhere between the 10th week (40 mm. stage) and the 20th week (125 mm. stage) of embryonic life.

The footplate was present in this case, but was likewise small and thick. Fixation was apparently due to bony fusion to the margins of the oval window. This was disrupted by mobilization maneuvers at the head of the stapes.

Without the necessity of removing bone from the external canal wall, the pyramidal eminence is well seen, as is the bony ridge of the facial canal. The pyramidal eminence is a continuation of the facial ridge in this ear. This position and amount of projection is considered very unusual.

Total absence of the stapedius muscle will be seen in Figure 5. This ear, however, has an extremely short, thick tendon. It was so short that I first believed the stapes to be joined directly to the pyramidal eminence with bony union. It was only after mobilization that I could see tendon motion.

Figure 5.

Photograph of a left middle ear displayed for the purpose of demonstrating muscular and bony malformations.

This ear is an example of total absence of the stapedius muscle, tendon, and its pyramidal eminence. This is a deformity without evidence of other known head or neck malformations. It was accompanied by fixation of the footplate of the stapes. The problems encountered during the operation were no different from those seen in any other ear with ankylosis of the footplate margin.

Much has been written concerning the function of the stapedius muscle. Five ears with congenital absence of this muscle are reported in this series. In four of these patients, serviceable hearing was obtained by operative restoration of mobility of the ossicular chain. In none of the four cases were there symptoms which one might expect from either the loss of the so-called "protective mechanism" or the lack of the so-called "tuning-up ability," which has been ascribed to the stapedius muscle by some authorities. This does not refute the theories of function of the stapedius muscle, but it establishes the fact that this muscle is not absolutely necessary for serviceable hearing. Please see the pre- and post-operative audiogram of this patient following the discussion of Figure 28.

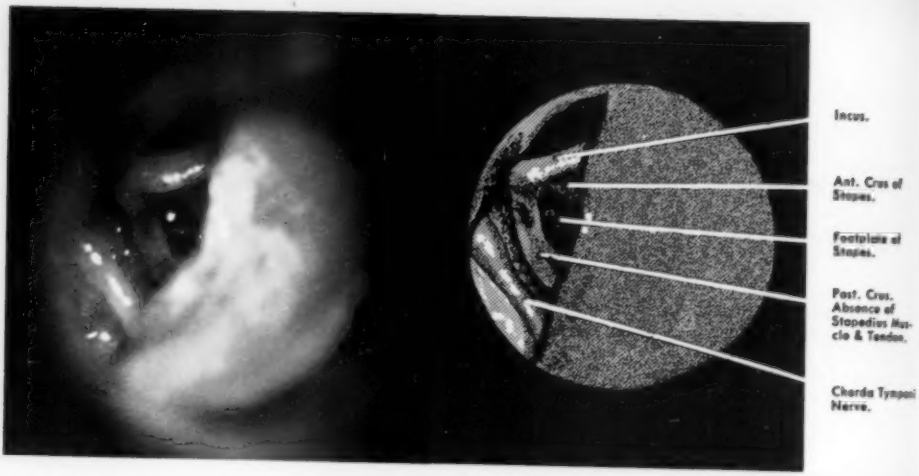


Figure 6.

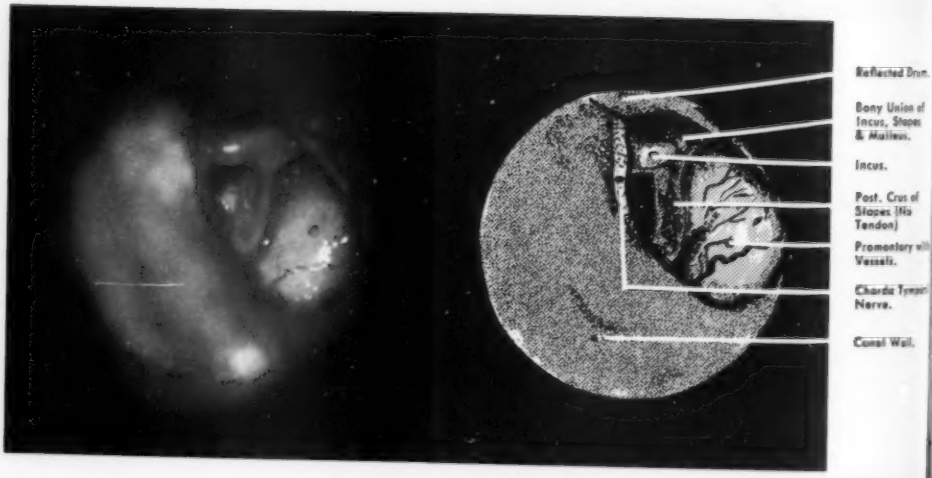


Figure 7.

Figure 6.

Photograph of a left middle ear displayed for the purpose of showing bony and muscular malformations.

In this ear there was a bony union of the promontory to the inferior surfaces of the crura of the stapes. There was a tremendous amount of longitudinal overgrowth of the long process of the incus.

At operation, the crura of the stapes were gradually separated from the promontory and the stapes was made mobile. For fear of possible re-attachment to the promontory, a repositioning of the incudostapedial joint was attempted. The joint was purposely severed and the long process of the incus was shortened. The head of the stapes was lifted superiorly so that its crura would not be against the promontory. The head of the stapes was again made to rest on the inferior surface of the long process of the incus. This questionable procedure proved to be worthwhile as shown by the ultimate functional result obtained. Please see the pre- and post-operative audiograms reproduced following the discussion of Figure 30.

The absence of the stapedius muscle, tendon, and its pyramidal eminence is again seen in this ear.

Figure 7.

Photograph of a right middle ear displayed for the purpose of showing bony malformations. This ear also has a muscular abnormality and a variation in the position of the round window is seen.

The rare malformation of triple bony union of the malleus, incus, and stapes is more clearly seen in Figure 8 and will be discussed there.

The posterior crus of this stapes is clearly seen and is without its normal arch. It is straight, but bends in a superior direction as it joins the footplate. The posterior crus is not only without its normal arch but also is longer than the anterior crus.

There is a total absence of the stapedius muscle, tendon, and pyramidal eminence in this ear. This is one of five cases in this series with this abnormality. To my knowledge, this abnormality has not been reported in ears without severe accompanying deformities. The embryologic considerations will be given in the discussion of Figure 8.

The footplate of the stapes is clearly seen just under the posterior crus. The anterior crus of the stapes is hidden by the long process of the incus.

Round window variations will be demonstrated in figures to follow, but an unusual variation in the position of the round window is here seen. In this ear, the round window is almost completely hidden by the overhang of the bony external canal wall and is placed far posteriorly. Without searching, one might be misled into believing that there is a total absence of the round window in this patient.

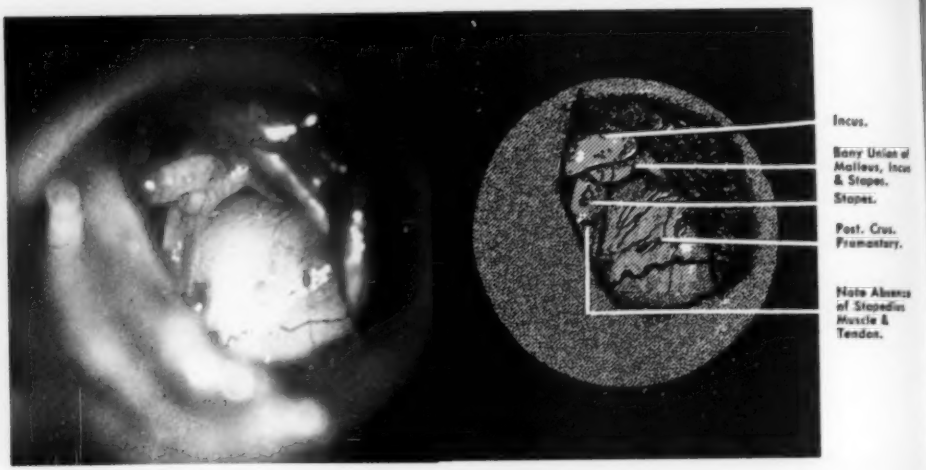


Figure 8.

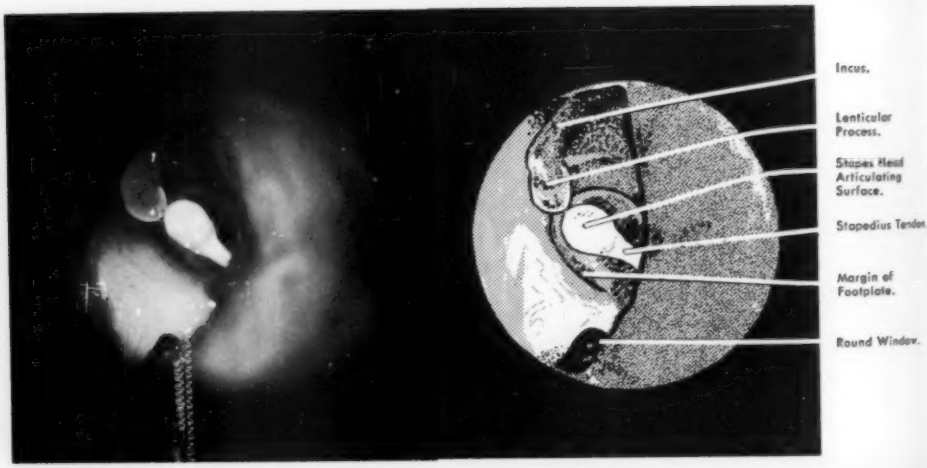


Figure 9.

Figure 8.

Photograph of the same ear as seen in Figure 7 presented for the purpose of demonstrating more clearly the bony malformations.

This ear, as previously stated, represents a rare triple bony union of the lenticular process of the incus with the handle of the malleus and the head of the stapes. This deformity, to my knowledge, has not been reported in the past. The long process of the incus is deformed, presenting a twisted S-shape. The handle of the malleus is clearly seen attached to the reflected tympanic membrane and is fused with the other two ossicles by a thick bony mass.

The problem of micro-orthopedics is obvious. Ossicular separation was accomplished and serviceable hearing was restored. The discussion of proposed embryologic significance of this deformity may be seen directly following the presentation of photographs.

Figure 9.

Photograph of a left tympanic cavity displayed for the purpose of showing a rare skeletal anomaly involving complete disarticulation of the incus from the stapes.

This patient suffered a severe blow upon his head during his youth and reported that partial loss of hearing in this ear began at that time. No other symptoms are present and the opposite ear is functionally normal.

Ossicular disarticulations due to nonfatal blows on the head have not been reported in the literature available to me. One would, therefore, hold some reservation in stating that trauma is definitely the etiologic factor, but due to the history it seems to be the most likely cause.

Consideration must be given to the possibility of this being a congenital abnormality. An embryologic explanation is difficult due to the absence of abnormalities in other structures closely related in development. The incus is derived from the first visceral arch, whereas the stapes is derived from the second visceral arch. During the 8th week (23 mm. stage), the lenticular process of the incus and the head of the stapes unite and a true articulating joint develops. A variation in growth and development possibly could produce a non-union of these bones as seen above.

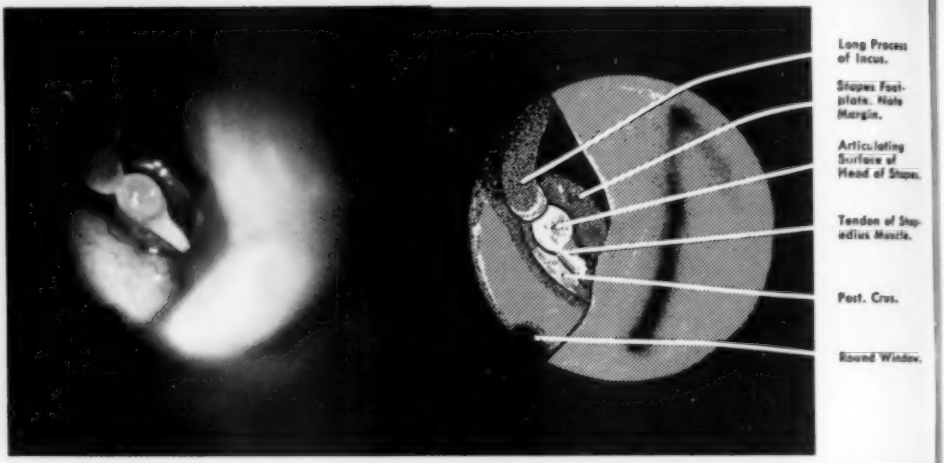


Figure 10.

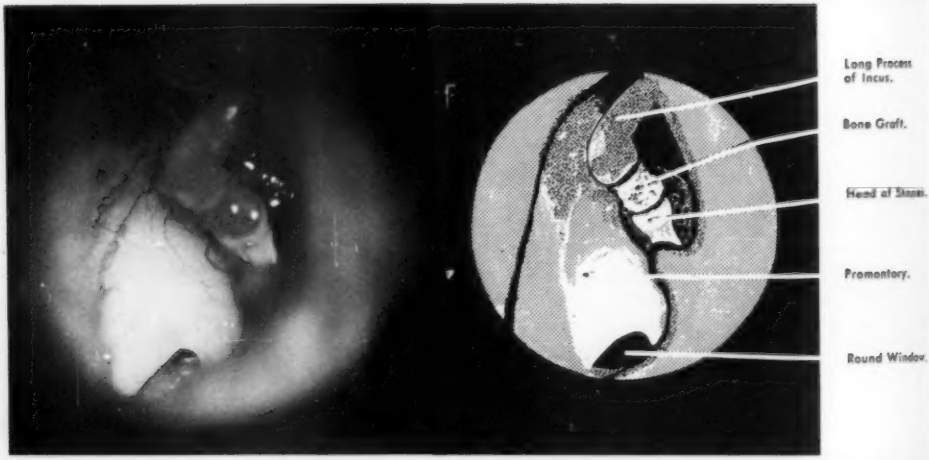


Figure 11.

Figure 10.

Photograph of a left middle ear (the same ear as reproduced in Figure 9). This brings the footplate of the stapes into better focus and demonstrates again the incudostapedial joint separation.

This patient had a unilateral purely conductive hearing loss of 67 decibels for pure tone and 62 decibels for speech. No other abnormalities of the middle ear could be found. The round window was present and the membrane visible. The malleus and incus were freely moveable. The entire stapes was easily seen. The footplate moved normally in the oval window. There was no evidence of abnormalities of the external or internal ear which would account for the profound loss of hearing. One may assume, with this evidence, that all of the hearing loss was due to disruption of the ossicular chain. Many observers have suggested that sound reaches the cochlea by molecular transmission through the round window in the theory of "hearing in reverse." Other investigators have suggested that the cochlea receives sound by a universal transmission of energy by all of the surrounding tissues and air without any point of emphasis. These theories deny the pre-eminence of transmission via the tympanic membrane and ossicular chain through the oval window to the cochlea. Some observers ascribe only protective function to the tympanic membrane and ossicular chain. It is not the purpose of this thesis to discuss theories of sound transmission, but the disarticulation presented in this patient caused consideration as to its functional significance.

This cochlea had the opportunity to receive sound transmitted through either the round window or the oval window. There was no mechanical barrier at either window. Both avenues of entry were perfectly normal; yet there was a loss of 67 decibels for pure tone. This type of hearing loss has been produced experimentally in animals by disruption of the ossicular chain continuity and testing cochlear potentials by the Wever-Bray phenomenon. This patient, however, represents a human example of the sound transmitting ability of the ossicular chain. Figure 11 will demonstrate a return of that function in the same patient by restoration of the ossicular chain.

Figure 11.

Photograph of the same left tympanic cavity as seen in Figures 9 and 10, displayed for the purpose of demonstrating operative correction of the incudostapedial joint separation previously seen.

The articulating surfaces of the head of the stapes and the lenticular process of the incus were roughened by the use of small picks. A small shaving of bone was removed from the edge of the bony external canal wall and teased into position between the articulating surfaces of the two ossicles. The hearing was instantly restored to normal and there was no regression in six months. Please see audiogram of this patient directly following the presentation of photographs.

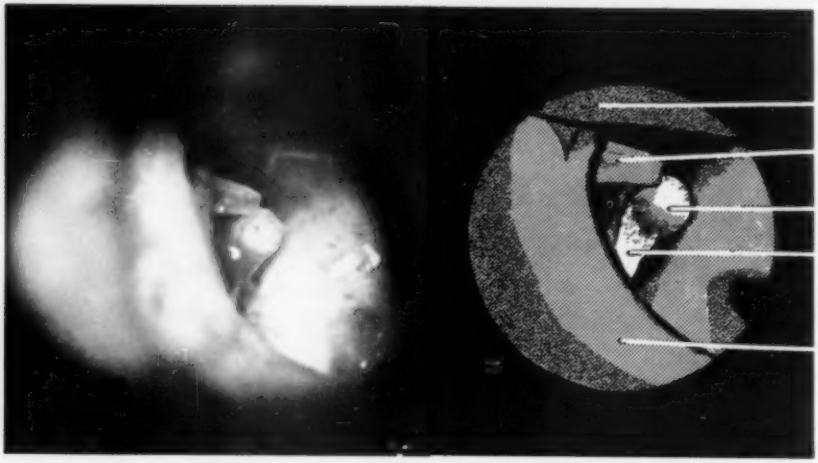


Figure 12.

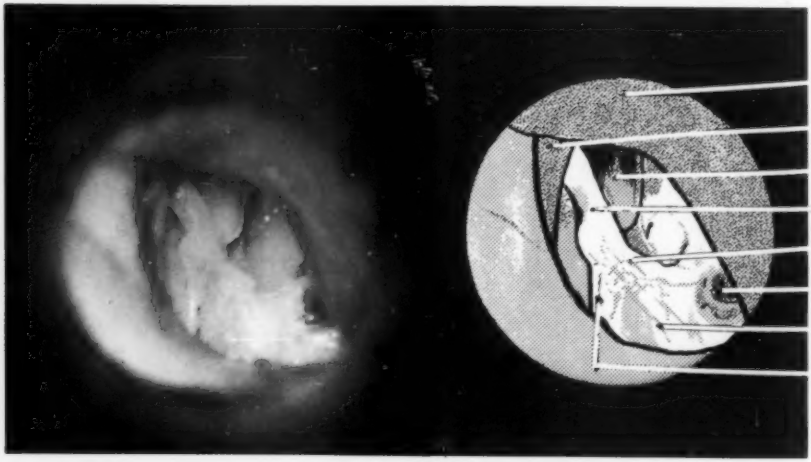


Figure 13.

Figure 12.

Photograph of a right middle ear displayed for the purpose of showing bony variations in the long process of the incus.

Frequently, the long process of the incus is the recipient of inflammatory destruction. This ear shows a total loss of the lenticular process of the incus; articulation with the head of the stapes is destroyed. There remains only a small strand of soft tissue between the incus and the stapes.

This type of necrosis is usually believed to be due to inflammation, but frequently this, or a variation of it, will be seen in an ear without history of suppuration. The tympanic membrane may be normal in appearance as it was in this ear. The ossicles may be normal in every other respect. There may be no evidence of adhesion in the middle ear and there may be no other evidence of previous middle ear disease. An early resolution of middle ear disease may have allowed necrosis of this vulnerable process and left the remainder of the middle ear undamaged. The hearing loss produced by this disruption of the ossicular chain is similar to that seen in Figures 9, 10, and 11.

Figure 13.

Photograph of a right middle ear displayed for the purpose of demonstrating an extreme bony malformation of the middle ear.

This ear belongs to a boy 10 years of age with a history of deafness since birth. The hearing loss was purely conductive in type (Please see the audiograms on the last page of this presentation.) There was no history of any intra-uterine etiologic factor and the family history was not illuminating. The external and internal ears were apparently normal. The tympanic membrane was normal in appearance and moved well.

The incus may be seen in this photograph to be placed far anteriorly. It has a lenticular process shaped like a golf stick directed almost completely posteriorly. The articulating face of this process seems to be directed posteriorly. This is perhaps better seen in Figure 14.

The bony ridge of the facial canal is many times its usual diameter and is placed extremely low in the middle ear.

A very large amount of unresolved primitive mesenchymal tissue tents over and covers in thick spider-web fashion the entire posterior superior portion of the middle ear. This obscured the anatomical landmarks and had to be removed (See Figure 14). This patient has no history of ear infections and this tissue is, therefore, not considered to be scar tissue.

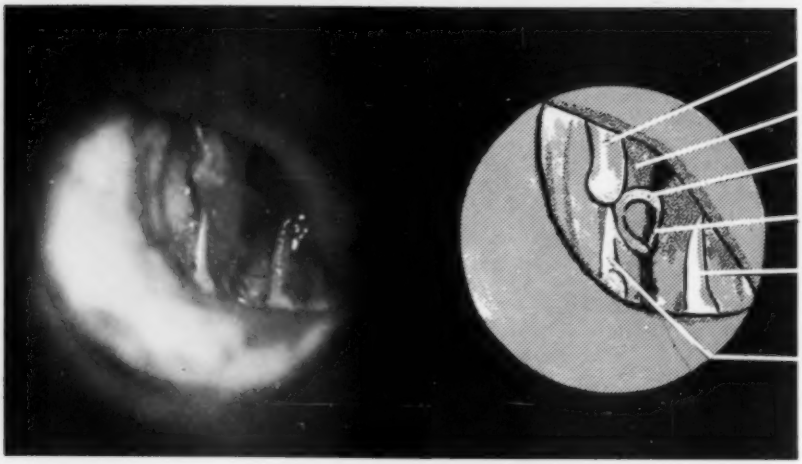


Figure 14.

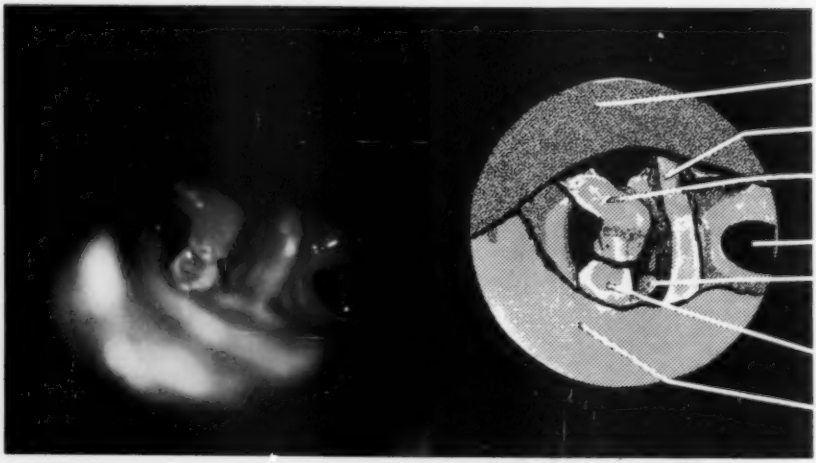


Figure 15.

Figure 14.

Photograph of a right tympanic cavity seen in Figure 13 after removal of mesenchymal tissue strands.

The two crura of the stapes can be seen wrapped around the facial canal like the claws of a bird on a limb. The crura are bony, but are extremely fragile. The are rounded off as they meet a very indefinite footplate. The oval window area is apparently recessed high up beneath the bony ridge of the facial nerve. There is a total absence of an oval window. A total absence of the oval window has been considered extremely rare. In literature available to me there were no human cases reported of this abnormality during the past 100 years. An attempt was made in this ear to find the oval window with the use of sharp probes, but only solid bone was encountered.

The stapedius tendon and pyramidal eminence are easily seen, but are very small.

A large bony spicule is seen projecting from the annulus to the promontory.

This malformation would have been overlooked and misdiagnosed without trans-aural tympanotomy. This patient's deafness has been consisdered to be due to a variety of conditions, such as hypertrophy of the tonsils and adenoids, Eustachian tube obstruction on the basis of allergy, adhesive otitis media, and congenital perceptive deafness. This patient had undergone tonsillectomy and adenoidectomy, radium application to the nasopharynx, Eustachian tube insufflation, and allergic management for the relief of deafness. The microscopic examination of this middle ear presented a correct diagnosis and initiated the planning of a rational surgical procedure for restoration of hearing.

Figure 15.

Photograph of a right tympanic cavity displayed for the purpose of demonstrating marked congenital malformations of the ossicles.

This is an ear of a 7 year old girl with a purely conductive deafness, present presumably since birth.

The incus appears fairly normal, but its body apparently placed more anteriorly than usual.

The head of the stapes has been purposely disarticulated and rotated posteriorly so that the posterior crus and the footplate area are in view.

There is a total absence of the anterior crus of the stapes in this ear. In animals, malformed unicurate, or columelliform, stapedes have been reported, but this is the first unicurate human stapes reported as far as I can determine, without accompanying external or inner ear deformities.

The footplate of the stapes is not present, but in its place is solid otic capsular bone. There is no oval window due to the lack of differentiation of the lamina stapedialis described in the embryologic discussion of Figures 13, 14, 15, and 16. The end of the posterior crus is clearly seen in the photograph. Audiometric findings of this patient may be seen directly following the presentation of photographs.

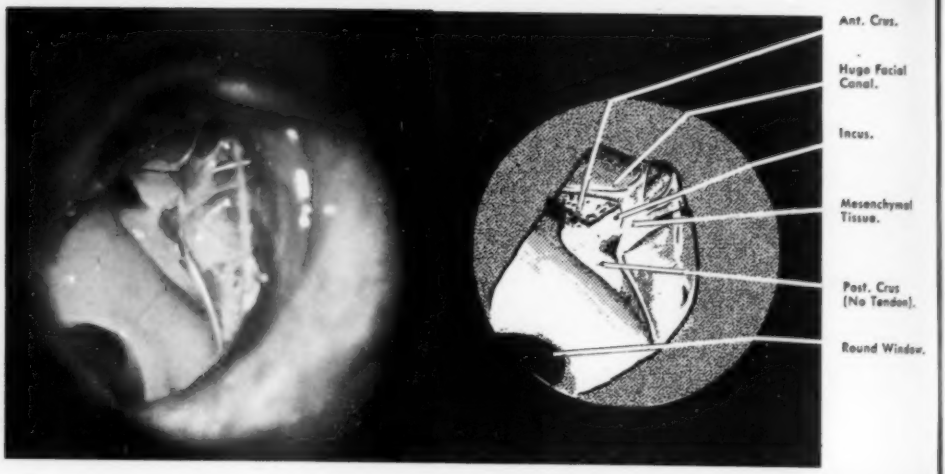


Figure 16.

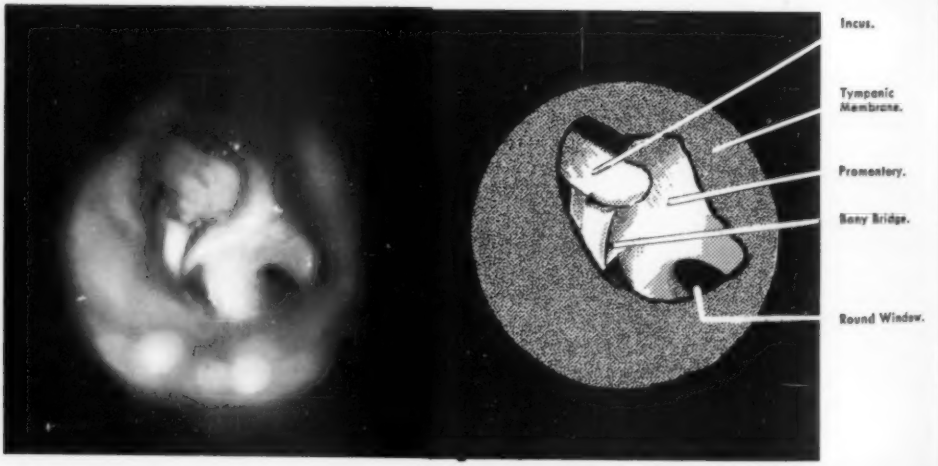


Figure 17.

Figure 16.

Photograph of a left tympanic cavity displayed to show marked bony malformation and absence of the stapedius muscle and tendon.

This is another ear representing a congenital conductive deafness without accompanying external or internal ear malformations.

There is a total absence of the stapedius muscle, tendon, and pyramidal eminence.

The long process of the incus articulates with the poorly developed head of the stapes. The anterior and posterior crura of the stapes are easily seen disappearing into a very narrow niche between the bulge of the bony facial canal and the promontory. The crura are very small and end as free stumps in the area of the oval window.

This is the third ear with complete congenital absence of the oval window reported in this series. There is no evidence in this ear of differentiation of the lamina stapedialis and the surrounding capsular area into a footplate for the stapes or an oval window.

The report of these cases lends evidence to the theory that the stapes footplate is derived from both the second visceral arch and the otic capsule.

The bony canal for the facial nerve is extremely large in this ear and one can see strands of mesenchymal tissue superficial to it.

Trans-aural tympanotomy was necessary for a correct diagnosis in this patient. A bypass of the oval window area by the creation of another labyrinthine fenestra will be necessary in order to improve this patient's hearing.

Figure 17.

Photograph of a right middle ear displayed for the purpose of showing a bridge of bone.

A projection of a bony process from the promontory to the area of the facial nerve, or pyramidal eminence, is very common. This ear demonstrates, perhaps, a larger than usual example of this bony variation. This process is frequently so near the posterior crus of the stapes that bony union is considered to be present. It has been my experience that usually, as was true in this case, there will be no union, although sometimes this process makes a true bridge across this area.

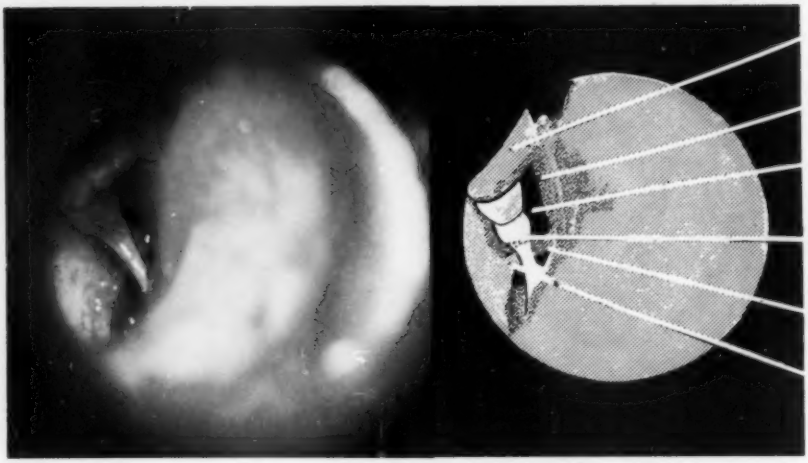


Figure 18.

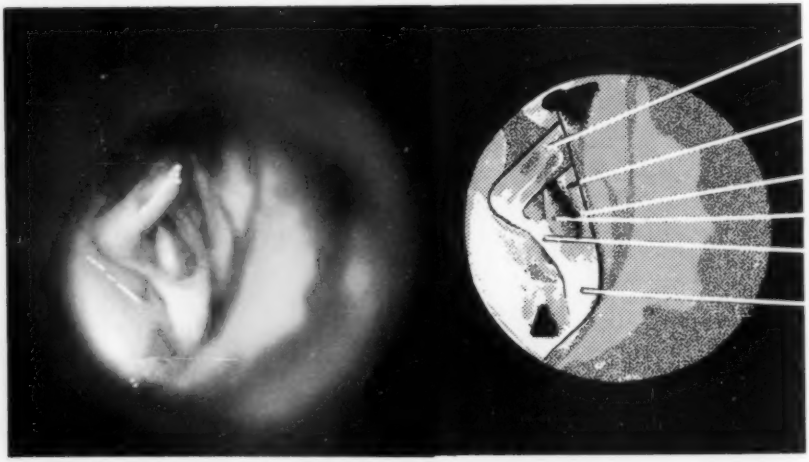


Figure 19.

Figure 18.

Photograph of a left middle ear displayed for the purpose of showing bridges of bone producing fixation of the stapes. Also seen is a narrow and deep niche for the stapes, and a stapedial head with an unusual shape.

A very high promontory, which bulges superiorly, has produced a very narrow deep niche for the stapes. A small bony process extends from the promontory to the posterior crus of the stapes. Another bridge of bone extends from the posterior crus to the bulge of the bony facial canal. Lysis of these bridges can be accomplished if good exposure can be obtained.

Figure 19.

Photograph of a left middle ear displayed for the purpose of showing a large amount of otosclerotic overgrowth. It also demonstrates an extremely short stapedius tendon, an exceptional view of the pyramidal eminence, and an unusual view of the ridge of the facial nerve canal.

In this ear, otosclerotic bone has completely filled the niche for the stapes. Masses of it have covered most of the crura so that only the head of the stapes seems to remain uncovered. The footplate and crural landmarks have been lost.

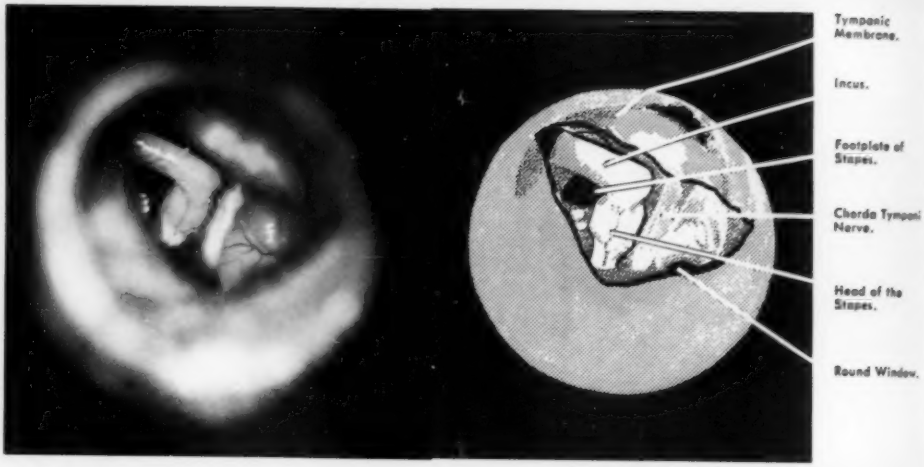


Figure 20.

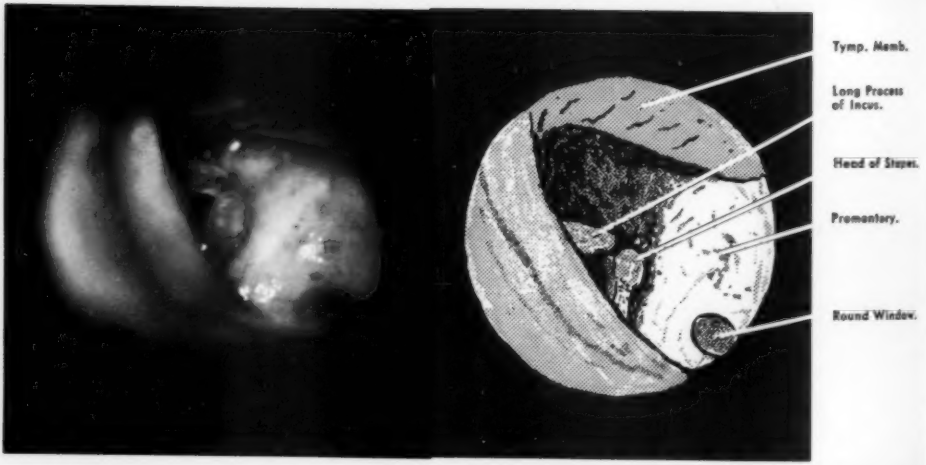


Figure 21.

Figure 20.

Photograph of a right tympanic cavity showing a usual view of the long process of the incus, head of the stapes, and footplate following the elevation of the chorda tympani nerve from its bed. This is shown for comparative purposes.

The incus is articulating with the head of the stapes in the most usual slightly anterior position. The lenticular process is well formed and appears normal. The long process of the incus is slightly curved medially and posteriorly and has no rough ridges on its lateral surface. The articulating surfaces are in a plane fairly square with the head of the stapes and footplate, permitting pressure, if necessary, through the incus on the stapes for mobilization purposes.

The amount of bony overhang of the canal seen in this ear is normal.

The footplate of the stapes is partially obscured by the thin cob-web strands of mesenchymal tissue. This is a common finding.

The tendon of the stapedius muscle inserts normally into the head of the stapes.

Figure 21.

Photograph of a right tympanic cavity displayed for the purpose of showing a variation in the long process of the incus.

This ear demonstrates an absence of the lenticular process of the incus, perhaps from necrosis. There is a noticeable lack of scar tissue in the middle ear. The tympanic membrane is normal in this ear and there was no past history of middle ear disease given by the patient. Nevertheless, one must consider the strong possibility of inflammatory disease producing such a picture.

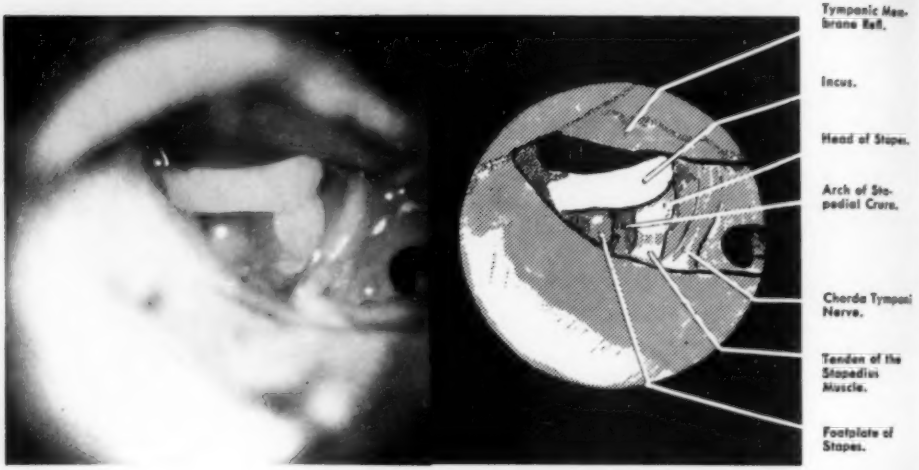


Figure 22.

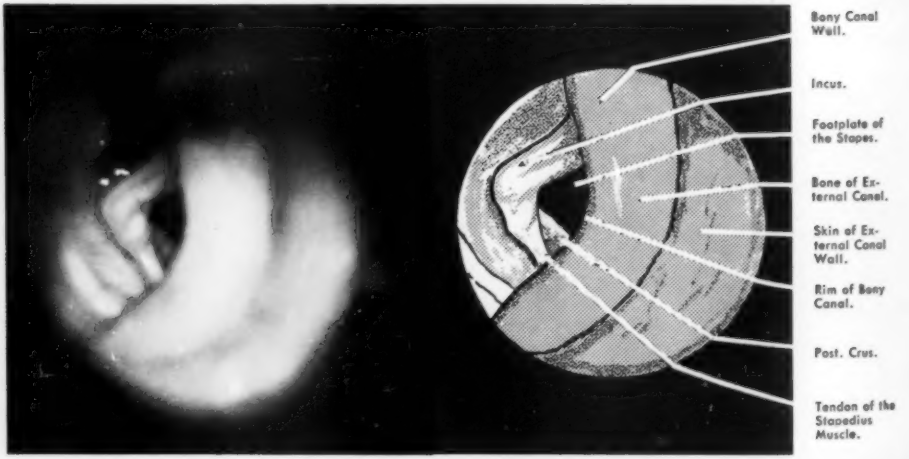


Figure 23.

Figure 22.

Photograph of a right middle ear presented for the purpose of showing variations in the long process of the incus.

The long process of the incus becomes visible at a lower level in this ear and points more directly inferiorly than usual. The most striking feature of this process of the incus is its length. The shaft is bowed, causing it to appear comma-shaped. It is turned upward at the end somewhat like the bow of a ship. Despite its length, it does not extend too far beyond the head of the stapes and its articulating surface even faces somewhat inferiorly. This articulating surface must be considered if pressure through the incus is to be exerted on the stapes for the purpose of mobilization. If the direction and pressure are faulty, dislocation is likely to occur.

The footplate of the stapes is clearly seen and is apparently infiltrated with otosclerotic bone. The crural arch of this stapes is extremely large.

Figure 23.

Photograph of a left tympanic cavity presented for the purpose of demonstrating variations.

The long process of the incus is turned medially at its tip and is rounded at this extremity. Articulation with the head of the stapes is far anterior, but the articulating surface is tilted only slightly anteriorly.

The tendon of the stapedius muscle is inserted into the small neck of the stapes. These findings are not considered unusual but are variations within the range of normal.

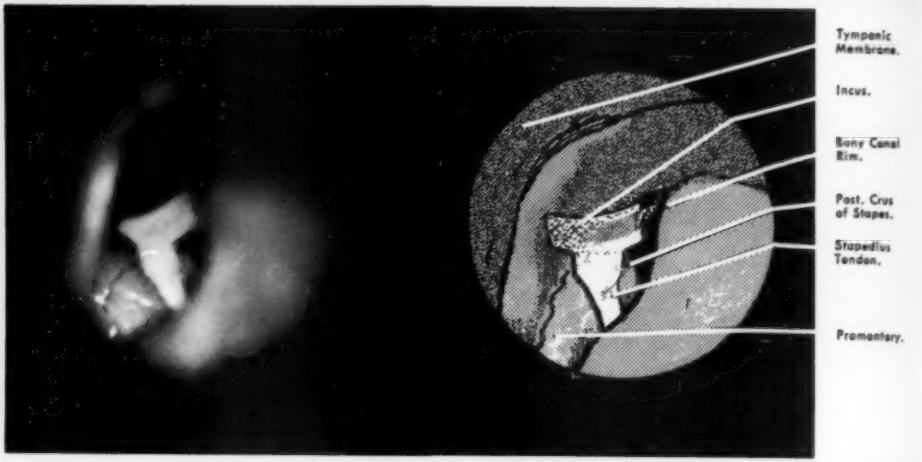


Figure 24.

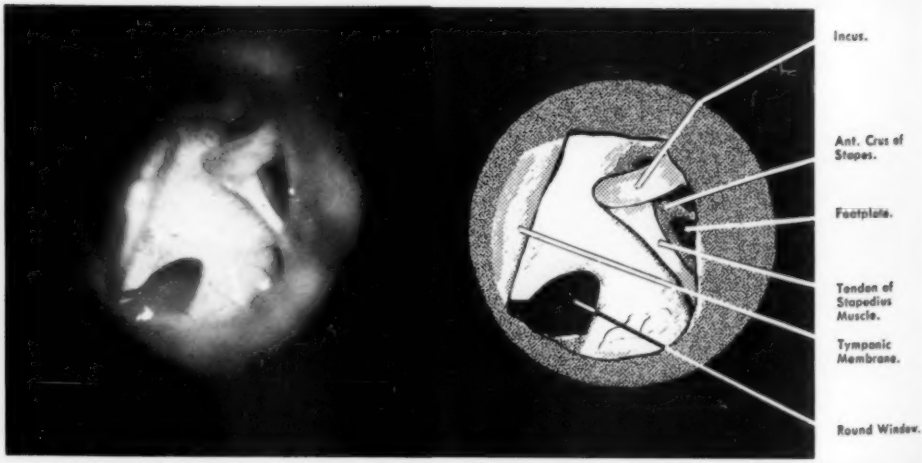


Figure 25.

Figure 24.

Photograph of a left tympanic cavity presented for the purpose of demonstrating variations in the long process of the incus.

The long process of the incus has a sharp bony projection on its lateral surface in this ear. This turns up like the bow of a ship, and presents a rough lateral surface which must be considered if instrumentation is to be done in this area. The lenticular process of the incus articulates with the head of the stapes in its usual slightly anterior location. The stapedius tendon inserts into the head of the stapes from an inferior direction thus exposing the posterior crus.

Figure 25.

Photograph of a left tympanic cavity presented for the purpose of showing variations in the long process of the incus.

This view is not uncommon, but shows an uptilted long process of the incus in comparison to the slight medial downward turn seen in Figure 23.

The head of the stapes is placed far forward and the lenticular process of the incus is resting on its posterior aspect. The long process of the incus actually hides from view the head of the stapes. Please compare with Figure 24.

The stapes is tilted inferiorly and, therefore, the articulating surface is angulated. This position must be considered before pressure is attempted through the incus if a mobilization of the stapes is contemplated.

The stapedius tendon is exceptionally well seen without disturbance of the chorda tympani nerve or the bony external canal. The tendon inserts high on the head of the stapes in this ear.

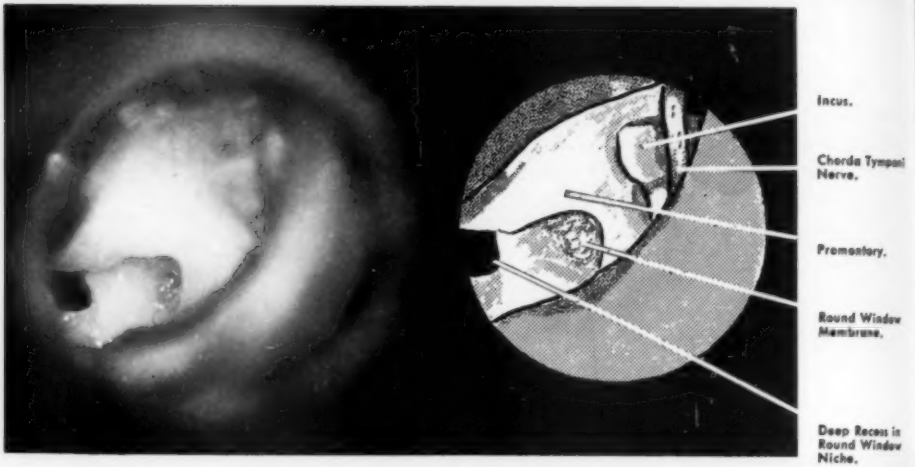


Figure 26.

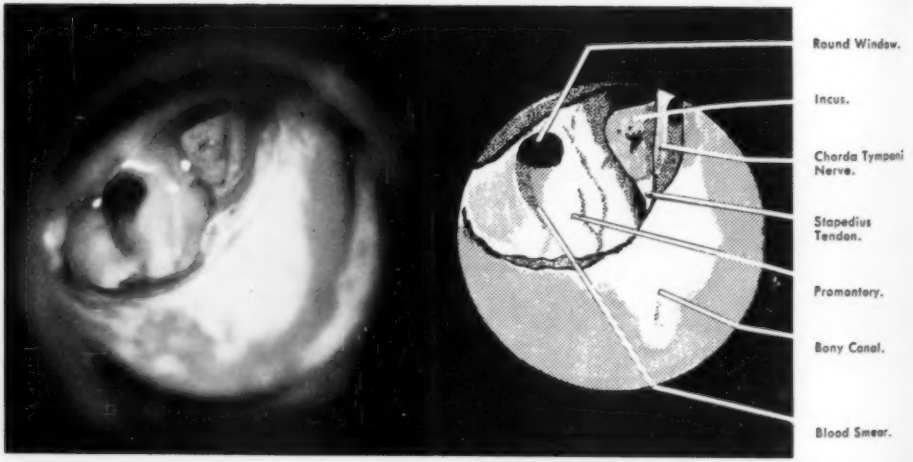


Figure 27.

Figure 26.

Photograph of a left middle ear displayed for the purpose of showing round window variations.

This round window membrane is seen clearly and is facing in the posterior, inferior and lateral direction. It is also on the usual posterior, inferior aspect of the promontory. A large wide niche is present and a very deep depression, or second window, is present at its inferior margin. I believe that most of the deep depressions commonly seen in this location are rarely in direct communication with the labyrinth.

Figure 27.

Photograph of a left middle ear displayed for the purpose of showing round window variations.

This round window is without a niche and is situated unusually far anteriorly. It is actually as far anterior as the oval window. The membrane is facing posteriorly, laterally, and slightly superiorly. The window is placed on the summit of the promontory.

This photograph also shows the usual view of the incus seen on lifting the tympanic membrane. The area hidden by the chorda tympani nerve, the moderate amount of bony overhang, and the barely visible stapedius tendon is considered to be average exposure of the middle ear.

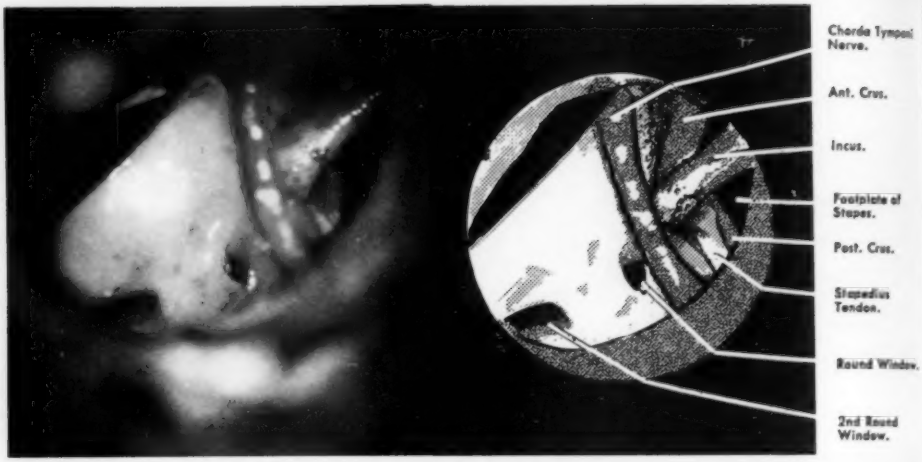


Figure 28.

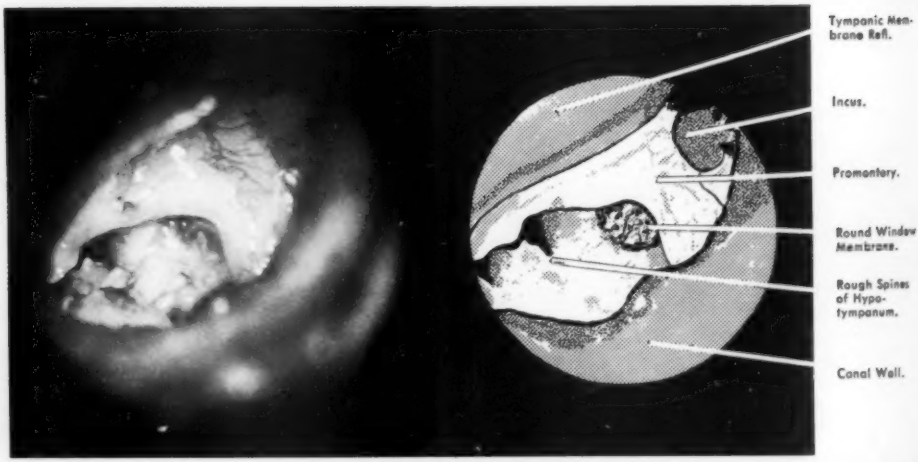


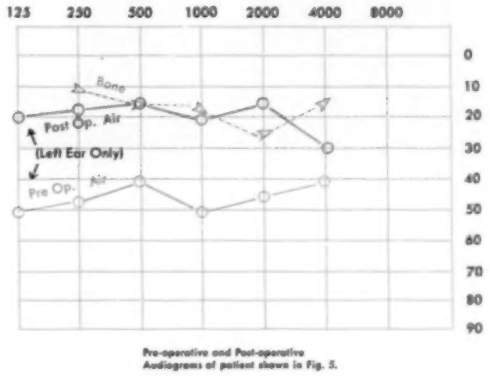
Figure 29.

Figure 28.

Photograph of a left middle ear displayed for the purpose of showing variations in the round window.

This is a view of two round windows which have no niche and are apparently completely separate. Membranes are thought to be present in both. The small superior window faces upward and laterally, whereas the inferior window faces posteriorly and inferiorly.

A large avascular promontory may be contrasted to Figure 31.



Pre-operative and Post-operative Audiograms of patient shown in Fig. 5.

Figure 29.

Photograph of a left middle ear displayed for the purpose of showing variations of the round window.

A large round window niche is seen in this ear and multiple rough projecting bony spines are seen inferiorly. These are frequently seen in the hypotympanum.

The large round window membrane is clearly seen covering the fluid of the labyrinth. The membrane is facing laterally, inferiorly, and posteriorly. This position on the posterior inferior aspect of the promontory is the usual position for the round window.

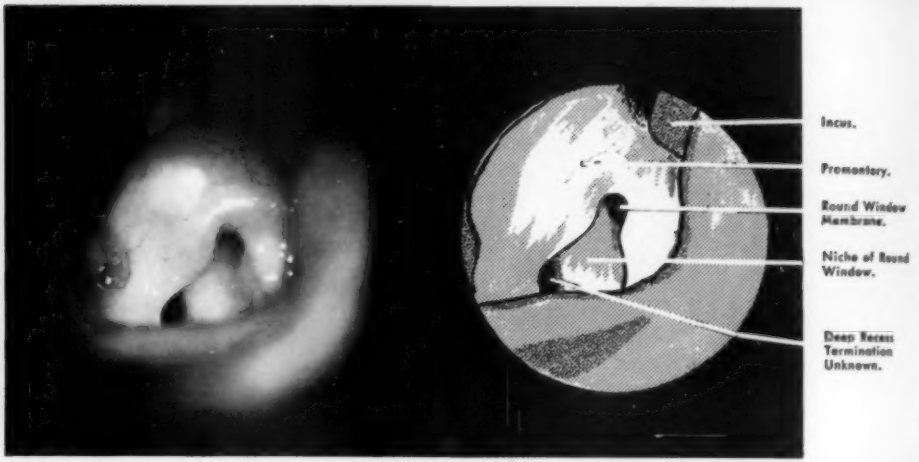


Figure 30.

Figure 31.

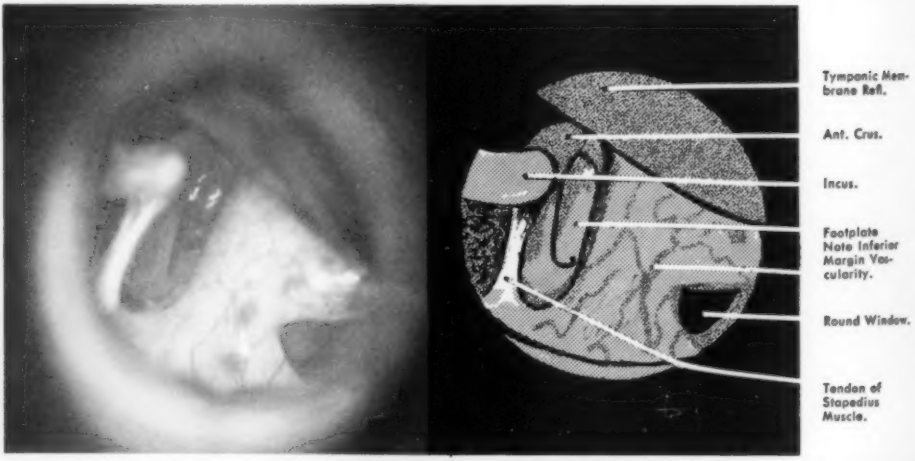
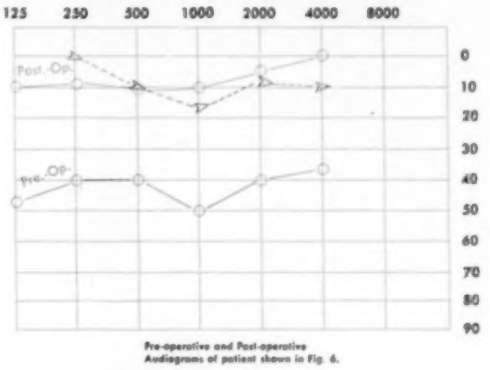


Figure 30.

Photograph of a left middle ear displayed for the purpose of showing variations in the round window.

This could be called the double-barreled round window. The upper window has a definite membrane which is extremely small and faces inferiorly and slightly laterally. The lower window may, or may not, have a definite labyrinthine connection with a separating membrane. These two depressions are located in a well defined niche which does not merge into the hypotympanic recess inferiorly as was seen in Figure 29.



Pre-operative and Post-operative Audiograms of patient shown in Fig. 6.

Figure 31.

Photograph of a right middle ear displayed for the purpose of showing variations in the round window, the stapedius tendon, the stapes, and vascularity.

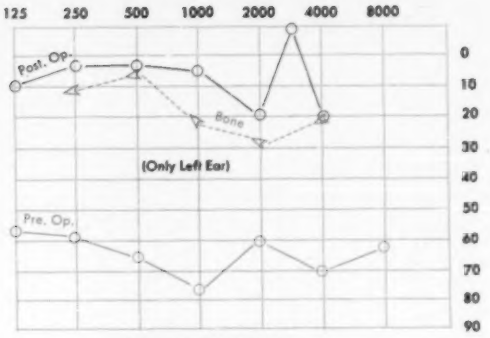
This is a very large round window and is situated in the usual promontory position but is without a niche. The membrane of this window is facing mostly in a lateral direction and is unusually superficial.

The stapedius tendon is quite long and is attached directly to the head of the stapes. It is well seen in its entirety, including the pyramidal eminence. This is unusual unless a portion of the external bony canal wall has been first removed. The stapedius tendon is also directed slightly superiorly. This is not too unusual, but is still in its most infrequent position.

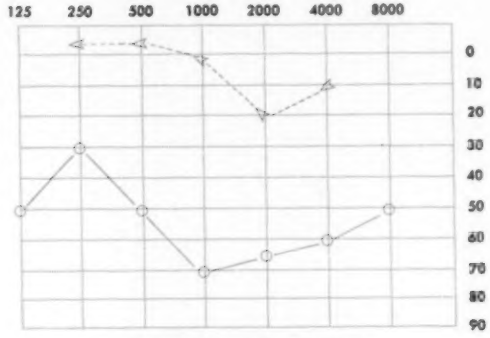
The stapes is located in a very shallow niche and its crura are turned slightly inferiorly. An unusual view of the entire stapes is seen. The crura are unusual in that they are approximately the same length and are similarly arched. A rare view of the entire lower margin of the footplate is seen.

The incus articulates with the head of the stapes in the more infrequent posterior position.

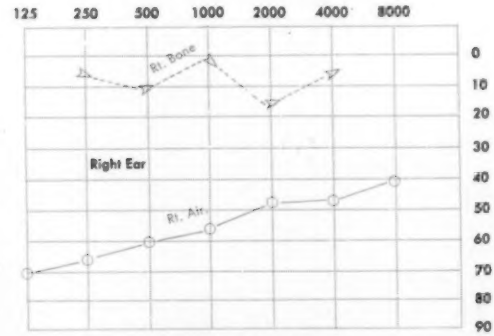
More vascularity than usual is present in this ear. The amount of vascularity is extremely variable and this ear does not represent the most extreme vascularity that may be seen. Note the large number of vessels over the promontory and the blood-filled appearance of the crura. This may be compared to Figure 30.



Pre-operative and Post-operative
Audiograms of patient shown in Figs. 9, 10, 11.



Audiogram of patient shown in Figs. 13, and 14.



Audiogram of patient shown in Fig. 15.

observed in numerous places in the bony orifice of the round window (see Figs. 29, 22). In a majority of patients, what appeared to be residual strands of mesenchymal tissue formed bizarre patterns over the window (see Fig. 29).

The usual round window could be seen easily immediately upon opening the middle ear in about the 8 or 9 o'clock position and on the inferior posterior slope of the promontory (see Fig. 26). The round window membrane is usually seen clearly, and is facing posteriorly, inferiorly and laterally. It is seen to move with good amplitude on normal footplate motion. The round window may be seen high up on the promontory in a very anterior position, with the membrane facing in a superior, posterior, and lateral direction (see Fig. 27), or it may be seen located far posteriorly so that it is almost entirely hidden by the external canal wall (see Fig. 20). An inferior 7 o'clock position is also possible.

The round window usually is located in the superior anterior end of a wide, long, excavated bony niche (see Fig. 26). This niche usually is roughly bean-shaped and may have bony projections or spines in the lower end (see Fig. 29). This may be continuous, with the same rough bony topography frequently seen in the hypotympanum. The lower end of this niche may, on the other hand, be smooth and present another rounded depression, or tunnel, which gives the appearance of being a second round window (see Fig. 30). Dissection to find the termination of some of these so-called double windows has not been possible, but this finding is frequent enough that postmortem investigation may soon give further light.

Many ears are seen in which there is no niche for the round window (see Fig. 28). The labyrinth tunnel orifice ends without elaboration on the promontory in a variety of positions as described above. The true round window membrane almost invariably, however, is recessed a short distance in this tunnel. Superficial tympanic mucous membrane may bridge over the exterior of the orifice, but investigation usually reveals that this is a pseudo round window membrane, and the true round window membrane will be found in a deeper position.

EMBRYOLOGIC DISCUSSION OF FIGURES 7 AND 8.

The stapes is derived from the second visceral (branchial) arch, and even after differentiation is well under way (16 mm. stage), the head of the stapes is still attached to the original branchial arch. This remaining connection eventually attaches to the otic capsule and becomes the pyramidal eminence and the stapedius muscle and tendon. One would assume that premature separation of the stapes from the second visceral arch occurred in this ear; therefore, total absence of the muscle is observed and no trace of its origin or insertion can be found.

The malleus and incus are derived from the first branchial arch and appear first at the 13 mm. stage as one mass. By the 25 mm. stage, the malleus and incus are easily identified and are separated by a distinct incudomalleolar joint. It is about this time that the long process of the incus gradually nears and finally joins the head of the stapes in an articulation. The triple bony union, seen in Fig. 8, represents disturbed differentiation during this development. Since the connection of the head of the stapes, with its second visceral arch, detaches and joins the otic capsule to become the stapedius muscle; one might expect it occasionally to be misdirected and to join the handle of the malleus. This would explain the triple bony union seen, but this explanation would also require that tissue differentiation be directed into bone instead of muscle.

Improper development of the first branchial arch in the formation of the malleus and incus is another possibility. These two bones are at first one mass and it is conceivable that an isolated area in the region of the long process of the incus and the handle of the malleus might remain fused and eventually unite with the stapes.

The normal attraction of the stapes head for the incus might also be duplicated in the handle of the malleus; therefore, all three might fuse together in a triple union. The lack of articulating surfaces could be the result of immaturity.

EMBRYOLOGY.

The malformations seen in Figs. 13, 14, 15, and 16 are difficult to explain embryologically, due to the very thin fragile crura of the stapes. The immature stapes normally is large and bulky. All of the other variations would be explained by growth distortion beginning during the eighth to the twelfth embryonic week. By this time, the union of the incus and stapes has occurred, the stapedius muscle is attached, and the footplate of the stapes is beginning to be blended with the lamina stapedialis of the otic capsule. The process has not been completed in these patients, and the annular ligament has not differentiated to produce the oval window. As a result there is solid otic capsular bone throughout the region of the oval window in these ears.

DIAGNOSIS.

In all the cases specifically discussed in this thesis the external canals and tympanic membranes were normal. The consideration of diagnosis is then in this light. One may, however, suspect a malformation of the middle ear in some patients prior to operation. The following observations may suggest middle ear anomalies: *1.* Cases of unilateral conductive deafness seem to have a higher percentage of abnormalities. *2.* Conductive deafness observed since birth or early childhood, either bilateral or unilateral, should suggest the possibility of middle ear abnormalities even in the presence of a normal tympanic membrane. Conductive deafness of this type is greater than that which would be due to any other acquired pathologic process.[73] *3.* The absence of Carhart's notch in the bone conduction curve may be of diagnostic significance.[73] A 50 to 65 db. air conduction loss is present in most patients, and both the air and bone conduction curves present a typical flat audiogram through speech frequencies.[73] In the patients reported here, Carhart's notch was present often, but the characteristic flatness of the air conduction curve was observed in every case. *4.* Congenital deformities of the middle ear should be anticipated if the patient demonstrates other anomalies. This is particularly true of deformities of the branchial arch derivatures. *5.* History dat-

ing back to accidental or operative injury must not be overlooked as a cause of middle ear damage producing conductive deafness.

In many instances, there will be no warning of significant variation or abnormality of the middle ear even after careful history, physical examination and audiologic tests have been employed. The use of a highly maneuverable operating microscope with direct lighting and magnification up to 40 diameters is necessary to study these interesting structural variations. One must be able to change both the position of the patient's head and the direction of vision through the microscope in order not to overlook the unusual changes in these minute structures. Insistence upon good binocular visualization and a routine of systematic inspection prepares the mind to attempt a rational surgical maneuver which will be best designed to fit the need.

ETIOLOGY.

The word variation is used here in the sense that it is an all-inclusive term indicating any deviation from normal. It, then, includes a vast variety of conditions. In this sense, variations seen in the middle ear may be either congenital or acquired in origin.

CONGENITAL DEFORMITIES.

Congenital deformities may be due to disturbed embryologic development on the basis of hereditary defects. Examples of this are the Treacher-Collins[154] syndrome, or severe varieties of otocephaly.[7,68,133,159] These cases do not fall in the group of isolated middle ear defects.[7] These malformations usually involve several of the branchial derivatives.[77] In all of the available literature I could find no single case of middle ear deformity without other accompanying deformities in which there was a definite family history; furthermore, in the cases reported in this thesis, there were no histories of similar conditions being present in other members of the family. In any of the more major deformities seen in the middle ear which do not have an obvious origin, we must

logically, however, consider heredity to be a strong etiologic possibility.

Congenital deformities may be due to intra-uterine fetal injuries. Deformities in certain cases have been ascribed to maternal viral disease, such as mumps, rubella[139] and poliomyelitis.[138,61,83] Fetal position in utero has been considered to be a factor in the production of congenital deformities. One investigator reported changes in the inner ear and central nervous system in the newborn with abnormal fetal positions.[119] He examined six patients with abnormal position and five of the six had histologic changes. In two, changes were found in the inner ear, and in one there was a severe disturbance in the development of the inner ear and middle ear. Collaboration of investigators with obstetricians might in the future shed light on this suggestion. All of the cases found in the literature in the above categories had multiple defects outside the middle ear. None of the cases reported in this thesis have known accompanying deformities outside the middle ear, and none of the conditions reported here could be traced directly to intra-uterine injury as a possible etiologic factor.

Variations in the middle ear may be produced by congenital deformities due to alteration of developmental processes by a variety of unknown causes before birth. The vast majority of variations have no obvious cause. Tardy or delayed resorption of mesenchyme in the middle ear has been considered to be due to hormonal or enzymatic imbalance.[63] If hormonal and enzymatic factors are effective in altering the resorption of this tissue, it is reasonable to assume that these factors might affect other developmental processes. Diabetes,[82] radiation,[81] dietary deficiencies, incompatible blood types[84] (RH factors), eclampsias, maternal and fetal hypoxia,[85] and excessive use of drugs might conceivably produce changes in the normal pattern of development. An unusually high incidence of abnormalities of the ear occurs in persons born prematurely. The conditions listed above are only presumptive possibilities, and thus far there has not been a well documented case reported in which these factors have been proven to cause isolated middle ear malformations.

Middle ear variations may be due to Mendelian recessive characteristics. The suggestion of this as a separate etiologic factor may be considered unreasonable. Actually, I have encountered and reported here several separate and distinct variations, such as total absence of the stapedius muscle, tendon and pyramidal eminence, which could conceivably fall in this category. These cases are unrelated to any other known embryologic developmental anomaly in these individuals. An embryologic explanation is difficult. In assigning these cases to a deviation at any particular time from the pattern of normal histogenesis, one finds difficulty in explaining why other closely related structures in the same patient are normal. The total absence of one separate part of a circumscribed area in the branchial development is entirely different from most malformations of an embryologic nature. One other alternative of thought, not depending upon embryologic maldevelopment, is that of the presence of a structure developed under the genesis of a Mendelian recessive characteristic. This etiologic presumption answers many questions, but the lack of evidence leaves the launching ground of this theory untenable, at least for the present. Significant proof would require exhaustive genetic study of the patient's family. There would have to be definite demonstration of this variation, and the individuals displaying it would have to fall into a certain hereditary pattern. The example given (the absence of the stapedius muscle, tendon, and pyramidal eminence) occurred in three cases without associated anomalies; however, in two of these patients, the absence was unilateral. A great deal of work has been done on hereditary characteristics related to cochlear variations in deafness.[50] Due to the abundance of material available, this area of genetic research has far outstripped the minor and less frequent variations in the middle ear. It is hoped that lack of evidence will soon become less of a hindrance now that more operations on the middle ear are being performed.

ACQUIRED VARIATIONS IN THE MIDDLE EAR.

Injury during growth periods of the temporal bone could conceivably produce a variety of unusual conditions of the

middle ear. It is interesting to speculate on the possible cause of the total separation of the incus from the stapes head as shown in Fig. 9. The history of this case was suggestive of traumatic origin. At the age of 16 years, this patient received a heavy blow to the head, and he thought that this might have been the date of onset of his unilateral deafness. Needless to say, the adult memory of the details of a childhood injury is often cloudy, and the patient is likely to be influenced by suggestions given down through the years by professional people as well as by the immediate family. The dislocation of this joint by any nonfatal blow to the head is open to serious doubt.[141,143,80] As a matter of fact, to my knowledge, there has not been a case of incudostapedial joint separation reported in even those cases of crushing head injuries coming to autopsy.[80] I could find only one other case of incudostapedial joint separation in the literature.[150,72] This case was not elaborated upon and presumably was not due to head injury or inflammatory processes. The one patient presented here, however, gave an unsolicited history of head trauma and, unprompted, he correlated the trauma with the hearing loss. It may be possible that as more explorations of the middle ear are performed in the future we will encounter dislocations and malformations which have resulted from head injury.

There has been one case reported of incus dislocation.[142] This was found during an exploratory transmeatal operation, and was a case of total dislocation of the incus with subluxation into the middle ear. The dislocation had been produced by an injury which had occurred 14 years previously during a simple mastoidectomy. This case serves as a simple and important reminder that one should anticipate variations and disturbed anatomy in patients who have had previous operations on the ear.

Middle ear variations may be acquired due to pathologic processes. Stimulation of normal osteogenesis, or osteoporosis, by repeated vibratory trauma around the ossicles has been suggested.[52,97] In the one case reported,[97] there was a dehiscence in the dural plate over the epitympanum through which the head of the malleus projected into the middle cranial fossa. It was believed that vibratory irritation stimulated

osteogenesis, or osteoporosis, making possible the formation of bony projections and bridges which could fix the ossicle in place. It was thus suggested that irritation produced by vibratory activity in areas where bone is in contact with bone could be a stimulus promoting the formation of bony bridges. It has been my frequent observation that most stapedial crura rest against the promontory to some degree. These, with few exceptions, have no bony fusion, and yet the structures are so much in contact developmentally that the mucous membrane seems to make no attempt to grow between them, but rather appears to surround in certain areas the individual crus and the promontory as one unit. This proximity may remain for a lifetime without bony union. These facts cast considerable doubt on the theory of vibratory activity producing bony bridges. Reports of ossification of the stapedius tendon causing stapedial fixation have been made.[143] Bony bands extending from the promontory, or facial ridge, to the crus of the stapes, thus producing stapedial fixation, have been reported.[52] Vibrations and inflammatory irritations have been suggested as possible etiologic factors.[52,97] Attention has also been directed to the posibility of bony bridges (not otosclerotic) which could, in places, cross the annular ligament. The lack of proper differentiation from chondral tissue into a fibrous ligament could be responsible for stapes fixation in these ears. In this event, the primitive chondral tissue would develop into bone.

Infection with its pathologic sequelae is well known in the middle ear, and will be discussed only briefly in the light of these observations. Among the 500 cases considered in this thesis, there was an unusual lack of adhesions observed in the middle ear, even though a frequent history of childhood earaches and suppuration was obtained. This was an unexpected finding and points up the fantastic ability of the living cell to restore order out of chaos and purpose out of destruction. This ability may, however, serve to blind the otologist. The tympanic membrane may return to normal appearance and, in a few isolated cases, lead one to believe that all suppurative disease in the middle ear has also been resolved without an aftermath of scar tissue. In these cases, one may lift the

tympanic membrane only to uncover an extensive network of thick scar bands. True, scar bands should not be confused with the tiny strands of unresolved mesenchymal tissue which are so frequently seen around the malleus, the incus, and especially in the region of the stapes.[63] These strands are present to some degree in the majority of middle ears and apparently hamper sound transmission very little (see Fig. 20). In cases of delayed, or incomplete, development, this tissue may be much more in evidence (see Fig.13).

Atrophy and necrosis of bone, possibly due to infection, can be suspected in certain cases such as Fig. 21. Presumption that infection can produce such change is based upon the frequently seen erosion of the ossicles in chronic mastoiditis and chronic otitis media. The lenticular process of the incus seems to be a particularly vulnerable area of osteolytic attack. The total erosion of this end of the long process produces a separation of the incus from the stapes and results in a conductive deafness due to interruption of the ossicular chain. Isolated erosion, or atrophy, of this process separate from any other bony involvement is difficult to understand. Why the head of the stapes and the crura, or the malleolar head is not also eroded by the same pathologic process, is a valid query. Perhaps it is because the long process of the incus projects a greater distance from any base source of soft tissue blood supply than any part of the other ossicles. They may be the most vulnerable portion for inflammatory destruction. It thus may become the first part involved and, if the pathologic process is controlled, destruction occurs only in this area. Perhaps this deformity is common and is only now being seen.

Otosclerotic bone in this series could be seen in the great majority of cases. Foci of otosclerotic bone usually were seen near the anterior end of the footplate of the stapes. The amount of bony overgrowth varied from that which was barely distinguishable to large mounds which enveloped the base of the anterior crus of the stapes. Fairly frequently, otosclerotic bone was seen covering a portion of the footplate, or even infiltrating and distorting it in its entirety. Occasionally, the surrounding bony capsule of the labyrinth, the annular ligament, and the entire footplate could be seen to be

completely overrun by massive invasion of the pathologic process. In these cases it was impossible to distinguish the anatomical edge of the footplate, or indeed, to differentiate the footplate from the otic capsule. On rare occasion, the otosclerotic bone was seen in such huge mounds that it practically covered both crura and footplate, almost to the point of obliterating the obturator foramen of the stapes (see Fig. 19).

Many other diseases, such as diabetes, syphilis, tuberculosis, cholesteatoma, etc., can produce destructive changes in the middle ear. None of these was a factor in any of the cases reported in this series.

CONCLUSIONS.

In the past the discovery of middle ear malformations unassociated with other known defects has been extremely rare. A large number of variations and malformations of the middle ear have been observed by me in 500 trans-aural tympanotomies. It is reasonable, therefore, to assume that these are not rare.

Variations and malformations must be considered in operative procedures of the middle ear; otherwise, a slight distortion or absence of a landmark can produce a mechanical pitfall or an uncharted course for the surgeon.

Trans-aural tympanotomy in patients with unexplained deafness of a conductive type is a rational diagnostic, and sometimes corrective, procedure.

The study of variations and malformations leads to a better understanding of the physiology of sound conduction, and offers evidence to support or deny certain functional theories.

Investigative efforts in embryology, histology, genetics, pathology, and other basic sciences may be stimulated by the observation of malformations and variations in the middle ear.

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AMERICAN RHINOLOGIC SOCIETY.

The Annual Program Meeting of the American Rhinologic Society will be held at the Palmer House, Chicago, on Friday, October 17, and Saturday, October 18, 1958. The speakers will include Ralph Riggs, M.D., "Maxillary and Pre-Maxillary Approach to Septal Surgery"; Jules Masserman, M.D., "Relationship of Olfactory Factors in Neurosis in Animals". A paper on the "Physiology of Respiration" will be presented by David Cugell, M.D.; "Heat and Moisture Exchange of the Respiratory Mucous Membrane," by Irving Cramer, M.D.; "Second Golden Decade of Rhinologic Surgery—The Advances of the Past Ten Years," by Harvey Gunderson, M.D. A discussion entitled: "Concepts of Nasal Physiology as Related to Corrective Nasal Surgery," by Maurice H. Cottle, M.D.; "Bone Transplants" by Robert Ray, M.D.

There will be a panel discussion on Hump Removal, Roof Repair and Nasal Process Corrections.

The meeting will start at 1:30 p.m. on October 17 and adjourn at 4:00 p.m. on Saturday, October 18, 1958. For further details write Robert M. Hansen, M.D., Secretary, 1735 No. Wheeler Ave., Portland 12, Ore.

DEVELOPMENTAL ANATOMY OF THE TEMPORAL BONE AND AUDITORY OSSICLES IN RELATION TO SOME PROBLEMS IN ENDAURAL SURGERY.*†‡

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INTRODUCTION.

The bones of the human skull are developmentally dissimilar to a striking degree, each apparently following an individualized route to maturity: the frontal ossifies from two centers which appear in the eighth fetal week; the zygomatic from a single center, appearing in membrane bone in the second month; the sphenoid from 19 centers, the temporal bone from 14, the first one of which is formed in the fourth month; the last about five weeks later.

Most remarkable of all are the auditory ossicles, which are made over for new uses, transformed from branchial to

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†From the Department of Anatomy, Northwestern University Medical School and the Department of Anatomy, University of Wisconsin (contribution No. 629 from the former).

‡Various phases of the inclusive investigation, carried out under the auspices of the Central Bureau of Research of the American Otological Society, by the present authors and their colleagues at the University of Wisconsin, and at Northwestern University Medical School, have been reported in the following journals: Ann. Otol., Rhinol. and Laryngol., 51:343, 1942; 51:891, 1942; 53:42, 1944; 55:467, 1946; 55:278, 1946; 55:278, 1946; 55:700, 1946; 56:957, 1947; 57:103, 1948; 57:603, 1948; 58:739, 1949; 59:1088, 1950; 60:1072, 1951; 61:740, 1952; 62:1084, 1954; 63:394, 1954; 64:802, 1955. Arch. Otolaryngol., 10:459, 1929; 16:19, 1932; 18:1, 1933; 18:291, 1933; 23:509, 1936; 24:127, 1936; 27:402, 1938; 27:588, 1938; 28:676, 1938; 29:939, 1939; 30:183, 1939; 30:922, 1939; 32:771, 1940; 36:891, 1942; 37:650, 1948; 52:882, 1950. The Laryngoscope, 56:561, 1946; 66:785, 1956. Quart. Bull. Northwestern Univ. Med. School, 14:250, 1940; 14:258, 1940; 15:263, 1944; 18:33, 1944; 23:465, 1949; 25:96, 1951; 25:366, 1951; 26:344, 1952; 28:17, 1954; 29:21, 1955; 30:235, 1956; 30:331, 1956. Contributions to Embryology, Carnegie Institution, 21:53, 1930. Anat. Rec., 48:141, 1931; 59:15, 1934; 65:485, 1936; 99:55, 1947; 106:361, 1950. Proceedings of the School of Public Health, University of Michigan (Inservice Training Course, "The Acoustical Spectrum"), Feb., 1952.

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acoustic function. Quite as extraordinary as the way in which the separate elements of the auditory apparatus originate, grow and attain maturity, is the independence enjoyed by each as it follows a developmental course that finally leads to interdependent adulthood. Time tables here are as different from one another as the developmental history and adult condition of the parts when compared with corresponding features in the morphogenesis of a typical long bone.

OBSERVATIONS AND DISCUSSION.

1. Otic Capsule.

In the human embryo of five weeks (8 mm.), the auditory apparatus is represented merely by an epithelial vesicle which has lately been freed from the parental ectodermal layer (the future integument).

The surrounding mesenchyma is undifferentiated, there being as yet no histological prediction of change into precartilage for an otic capsule; however, no sooner has precartilage formed an encapsulating investment than deorganization of the newly-formed tissue begins around the parts of the endolymphatic (otic) labyrinth. A reticulum becomes vacuolated; then the vacuoles coalesce to produce the communicating spaces of the perilymphatic (periotic) labyrinth. These changes take place in a six-week period (between the fifth and the eleventh week).

In the fetus of nine weeks (40 mm.), both the otic (endolymphatic) and periotic (perilymphatic) labyrinth of the internal ear are formed; the otic capsule is a cartilaginous envelope for the labyrinthine ducts and surrounding canals and scalae. In the area of the future middle ear, the ossicles are formed in cartilage and lodged in mesenchyma. Growth in cartilage then continues through another period of six weeks.

The otic capsule of a fetus in the fifth fetal month (19½ weeks, 160 mm.) already displays distinguishing features in the cochlear and canalicular divisions: in the cochlear portion, ossification has been initiated; in the canalicular part, carti-

lage is not yet being replaced by bone; rather, it is being destroyed, rendered spongious to permit terminal increase in the arcs of the semicircular ducts.

The buds of vascular tissue that destroy the cartilage enter through the subarcuate fossa, which at this stage is a relatively ample excavation.

The first ossification center appears in the otic capsule at the age of about 16 weeks (115 to 120 mm.); the last (14th) at the end of the fifth month.

As soon as an ossification center is definitive, it is found to consist of three layers, each of which has its own rate of increment and maturation, and time of appearance in the cochlear and canalicular divisions of the capsule.

The inner periosteal (or endosteal) layer already forms a thin parietal shell for the turns of the cochlea; it is completed quickly but remains virtually unaltered throughout life.

An outer periosteal layer appears at the same time. Immediately other laminae are added externally—in steady acquisition, and after the regular pattern existing in the cambium layer of the periosteum in a typical long bone; however, the structure differs therefrom in that, despite growth, the simple laminated bone is never replaced by bone of Haversian type.

The middle layer, unlike the other two, is compound: it consists of a calcified cartilage matrix in whose lacunae invading osteoblasts have surrounded themselves with bone which, while formed in membrane, is deposited upon the framework offered by the calcified cartilage. This combination is intrachondrial bone. The intrachondrial tissue is formed concurrently with the outer layers of the ossification center, but production of the endochrondral bone is delayed until the early weeks of postnatal life, at which stage it is produced with sweeping rapidity.

The layers of the capsule are especially distinct in the fetus at midterm, *i.e.*, in a specimen of 21 weeks (183 mm.) in the

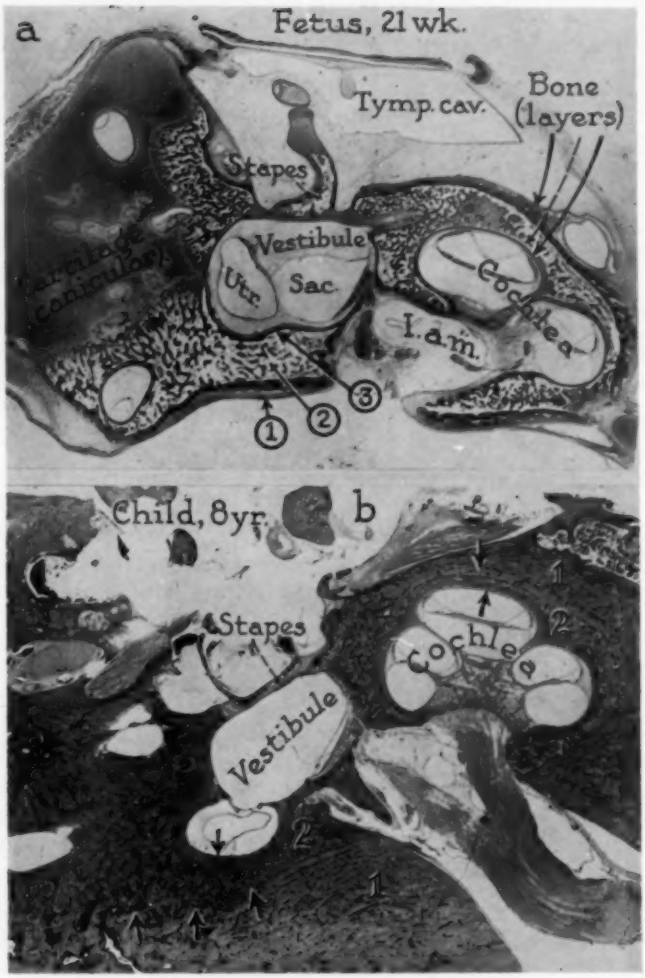

Fig. 1. Stages in the development of the otic capsule. Wisconsin series 21, and P37; a.—Fetus of 21 weeks (183 mm.); b.—Child, 8 years old.

Abbreviations: I.a.m., internal acoustic meatus; Sac., saccule; Tymp. cav., tympanic cavity; Utr., utricle.

Numerals indicate the layers of capsular bone. In the cochlear part of Fig. 1-a (to the reader's right) the downward-directed arrow points to the external limit of the middle layer; the upward-directed arrow to the internal (free) margin of the inner layer of bone. In the vestibular and canalicular divisions of the same figure (to the reader's left) the single arrow touches the internal margin of the inner layer; the three arrows point to the line of meeting of the outer and middle strata of bone.

cochlear, vestibular and canalicular portions of the capsule (see Fig. 1-a).

These layers retain their fetal appearance, even in areas where the capsule is being rebuilt. The tympanic wall of the lateral semicircular canal (the site of fenestration surgery) is one such area, and the external aperture of the vestibular aqueduct is another.

In a 38-week (350-mm.) fetus, cartilage persists in only two locations, *i.e.*, in the wall of the fissula ante fenestram and, continuous therewith, as a rim for the vestibular (oval) window. The outer (periosteal) layer has undergone considerable thickening; like the bone of the other two layers, it is non-Haversian and permanent. In the middle layer, the cartilage islands are covered by endochondral bone—marrow spaces being correspondingly reduced in bulk. In portions of the cochlear capsule, space is still a prominent feature; its existence serves to set off the outer from the inner layer. The inner layer is unchanged.

In the newborn infant the otic capsule is still cancellous in texture. This appearance is due to the histological structure of the middle layer, which is still composed mainly of intrachondrial bone. Because of its open texture, the layer is clearly distinguishable from the other two, the inner being relatively thin and smooth-surfaced. The outer layer will undergo steady increment until the temporal bone has attained adult form and dimensions. The middle layer will assume a petrous consistency as a result of the production of new bone (deposited upon the intrachondrial spicules). The inner layer will remain virtually unchanged throughout life.

In the ear of a child eight years of age, the three fundamental layers of the otic capsule are still distinguishable (see Fig. 1-b). The chief postnatal changes are two in number: increase in thickness of the outer layer; attainment of compact character by the middle layer. Frequently a large part of the middle layer will have been destroyed to make way for air cells. It has been established that pneumatization may be complete in the young child, and that in some individuals the process is under way in the late fetus.

2. Auditory Ossicles.

The structure of the stapes calls for special discussion in relation to problems presented to the surgeon in the operation for mobilization. Developmental history fully accounts for its architectural weakness.

In the fetus of 20 weeks (167 mm.) the single ossification center has made its appearance on the obturator wall in the area of continuity of the crus with the base.

At a slightly later stage (21 weeks, 190 mm.), an osseous shell is circumferential for the crus; otherwise, the ossicle remains cartilaginous. In the crural portion of the stapes, marrow has replaced cartilage.

In the 23-week (202-mm.) fetus, ossification has advanced to include the neck of the stapes, and to spread along the crura toward the head of the ossicle. Cartilage remains at the basal extremity, but is eroded internally; on the internal surface, endochondral bone is forming.

The stapes in the fetus of 24 weeks (215 mm.) is composed of bone, except at the extremities. Medially, cartilage covers the vestibular surface and the fenestral margin, whereas laterally the primordial cartilage of the capital end persists as an articular plate (at the incudostapedial joint).

The stapes of the 32-week (290-mm.) fetus has attained adult form and structure. It is completely invested by mucous membrane; both the basal and capital extremities are bilaminar (each of the layers being thin but complete).

All of the structural features which characterized the stapes in the late fetus and in the newborn infant are retained in virtually unaltered form in the adult: the ossicle is excessively thin; its base, like the head, is two-layered (the cartilaginous lamina of the vestibular surface investing the endochondral lamina of the tympanic aspect); mucous membrane not only covers the outer surface of the periosteal bone of the crura and head, but also coats the inner surface of the same layer where, as a result of removal of the obturator wall of the ossicle, the mucosal tunic comes to replace marrow

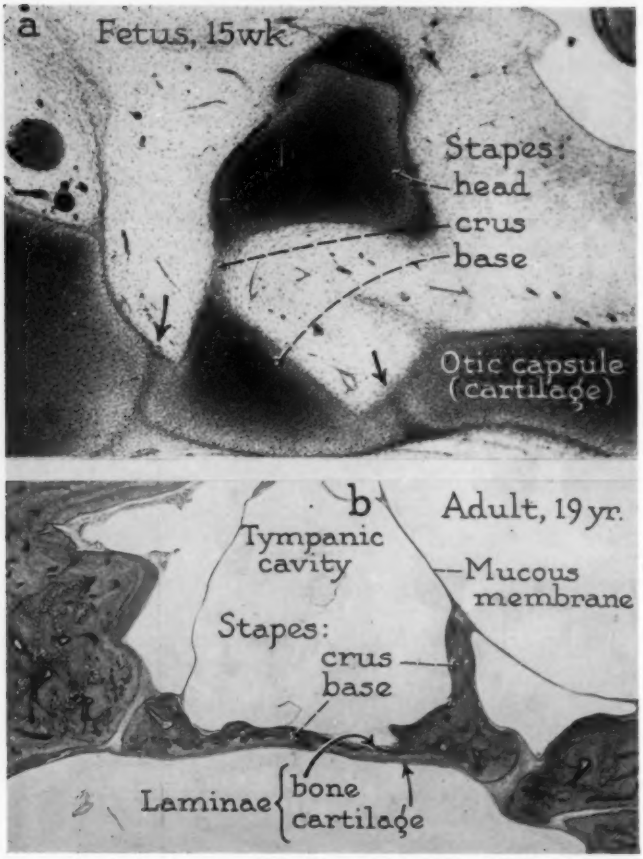

Fig. 2. Stages in the development of the stapes. Wis. 50 and P29. a.—Fetus of 15 weeks (111 mm.); b.—Adult 19 years old.

and endochondral spicules. The mucous membrane on the base of the stapes lies in contact with the endochondral lamina, which in a process of secondary growth, formed a covering for the persistent cartilage.

When once the stapes has been reduced in bulk and strength, it subsequently undergoes negligible alteration. The ossicle

remains infantile—or, rather, it is already an adult bone in the newborn.

By way of contrast, the histological structure of the incus is subject to remodelling at any age in a person's life span (see Figs. 3-a and 3-b); however, in some infantile specimens, excavation of both perichondral and endochondral bone may be under way, and in others, destruction is marked, but new bone is being applied to the walls of the recently formed

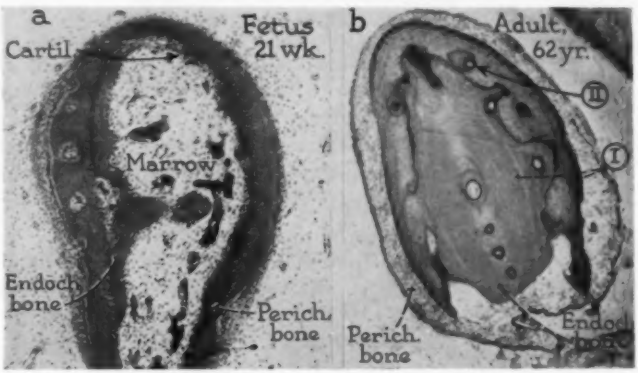

Fig. 3. Features in the development and adult remodelling of the incus (long crus). Wis. ser. 21 and P10. a.—Fetus of 21 weeks (183 mm.); b.—Adult 62 years old.

Abbreviation: Cartil., cartilage; Endoch. bone, endochondral bone; Perich. bone, perichondral bone. Encircled Roman numerals indicate primary (I), and secondary (II), ossification.

spaces. In some adults, erosion may affect chiefly the perichondral layer; in others, secondary excavations appear, as does concurrent formation of tertiary lamination.

CONCLUSIONS.

The otic capsule is already precartilaginous in the six-week embryo, whereupon the tissue begins to deorganize around the otic labyrinth, predicting periotic vacuolization (for semicircular canals, vestibule and cochlear scalae).

Although bone formation is under way in the cochlear

region at 20 weeks, the canalicular division not only remains cartilaginous for a four-week period, but is rendered spongious, permitting expansion of the semicircular ducts. Even when capsular ossification is virtually complete, the middle layer still consists mostly of intrachondrial bone. Soon after birth, rapid deposition of endochondral bone contributes petrous character to the entire capsule. The inner layer never changes; the outer thickens to imbed the capsule.

The capsule undergoes late remodelling at the external aperture of the vestibular aqueduct and along the tympanic wall of the lateral semicircular canal, while bone in the canalicular region is being pneumatized.

The ossicles, although derived from the branchial skeleton of the embryo, and ossified from a single center each, follow different courses of morphogenesis and at different rates of speed. The stapes of the midterm fetus has already attained adult dimensions, but is destined to undergo profound reduction in bulk before birth, after which it remains structurally quiescent. Like the otic capsule, the stapes never attains the Haversian stage of typical bone; in that respect it retains lifelong fetal structure. The incus, on the contrary, experiences remodelling of both its periosteal and endosteal layers throughout life, yet suffers no grossly discernible loss in bulk.

Difficulties which confront the surgeon in employing any one of the several techniques calculated to mobilize an ankylosed stapes are the result of the following circumstances: the anatomic position and relations of the stapes; its extreme fragility and its inability to heal, once it has been fractured; the power of the otic capsule to produce rapidly-growing bone in the territory of the vestibular (oval) window. All portions of the stapes are fragile; they are already frail in the infant, and will remain so throughout life. The crura and base are always thin, the head is pitted or deeply excavated, or both. The base (footplate) is usually the weakest part of the ossicle. In many cases, bone is a thin lamina on the tympanic aspect, *i.e.*, toward the middle ear; cartilage, persisting from the fetal stage, completes the base on the vestibular surface.

CONGENITAL STAPES FOOTPLATE FIXATION.

A Preliminary Report of Twenty-Three Operated Cases.*†‡

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and
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INTRODUCTION.

Otosclerosis has long been recognized as a cause of fixation of the footplate. We now realize there is a second cause, namely, congenital fixation of the stapes footplate. This entity does exist, can be diagnosed by clinical means, and can be recognized at the time of surgery.**

Shambaugh[1] has described five such cases, and Holmgren[2] reported a case of incomplete development of the oval window. Altmann[3] has described the histological findings of a case of atresia with congenital footplate fixation. Wolff[4] has observed abnormalities in the annular ligament of the new-born.

EMBRYOLOGY.

Embryologically, the malleus and incus are differentiated from the first branchial arch, or Meckel's cartilage. The stapes is derived from the second branchial arch, or Reichert's cartilage. Failure to differentiate results in a congenital abnormality of the structures involved; consequently, the stapes and stapes footplate may be involved without involvement of any of the other middle ear structures.

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†From the Department of Otolaryngology, University of Southern California School of Medicine.

‡Sponsored by the Los Angeles Foundation of Otology.

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DIAGNOSTIC FEATURES OF OTOSCLEROSIS IN THE ADULT.

The differential diagnosis between otosclerosis and congenital footplate fixation may at times be rather difficult. The key to the diagnosis of otosclerosis in the adult is *progression* of the hearing loss. If a patient has a slowly *progressive* conductive or mixed type of loss with essentially

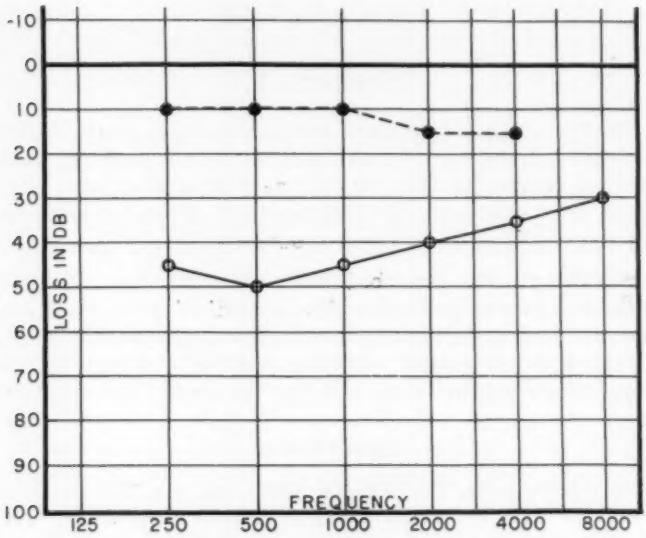


Fig. 1. Otosclerosis.

normal otologic findings, the diagnosis is usually clinical otosclerosis (see Fig. 1).

The amount of conductive impairment varies with the degree of otosclerotic footplate fixation. The air conduction curve usually shows the greatest loss in the lower frequencies. Cochlear involvement is quite common and usually involves the higher frequencies.

DIAGNOSTIC FEATURES OF CONGENITAL FOOTPLATE FIXATION IN THE ADULT.

The key to the diagnosis of congenital footplate fixation in the adult is the *absence of progression* of the hearing loss. A patient with a history of a purely conductive *non-progressive* hearing impairment, who has essentially normal otologic findings, may be diagnosed as ossicular discontinuity, or congenital ossicular fixation, occurring usually at the stapes footplate.

To determine accurately the *lack of progression* may be very difficult. These patients give a history of accidental discovery of the hearing impairment, either by school tests, or as the result of a certain incident that called attention to their hearing deficiency. The average age of such discovery in this series of cases was ten years. After discovery the patient, and parents, become suddenly aware of the impairment and proceed to test the hearing acuity in various ways. As the result one may feel that the hearing is getting worse, when actually the patient is only becoming more aware of the pre-existing deficiency. Because of the increased demands made on his hearing, one may feel the hearing loss is becoming greater as he gets older because of his more adult activities.

Patients with a congenital fixation of the stapes footplate have grown up with their impairment. They automatically compensate for their deficiency, and become excellent lip-readers long before their hearing loss is discovered. They frequently give a history of being slow in learning to talk, but all seem to have an excellent speech pattern (see Fig. 2).

Audiometric findings in congenital footplate fixation are remarkably constant. The air conduction curve is flat and averages 50 db. in the three speech frequencies. The bone conduction curve also is flat and remains within 10 db. of the zero threshold throughout the audiometric frequency range.

CONDUCTIVE LOSSES IN CHILDREN.

We recently reviewed our patients under 12 years of age who

had a conductive loss with essentially normal otologic findings, and who were examined in our office ten or more years ago. We found these cases could be classified into four groups.

GROUP NO. 1.

This group consisted of conductive losses of 15 to 35 db.

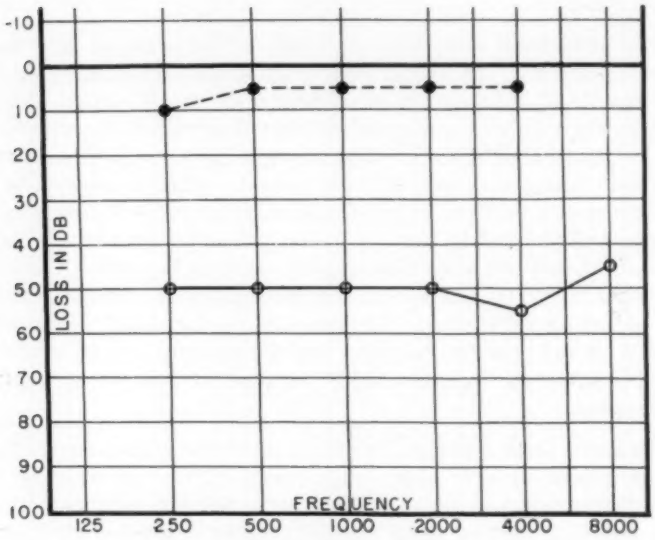


Fig. 2. Congenital Footplate Fixation.

in the three speech frequencies, which responded to therapy and had no further difficulty in adulthood.

CASE REPORT.

T. E., age five, was first examined in 1945 concerning a hearing impairment which was discovered by routine school audiometry. There was no history of middle ear disease, and the otologic examination was essentially normal. A tonsillectomy and adenoidectomy was performed, followed by X-ray therapy to the nasopharynx. The hearing improved following this therapy and has maintained the improvement to date, as per the following audiograms (see Figs. 3-A, 3-B).

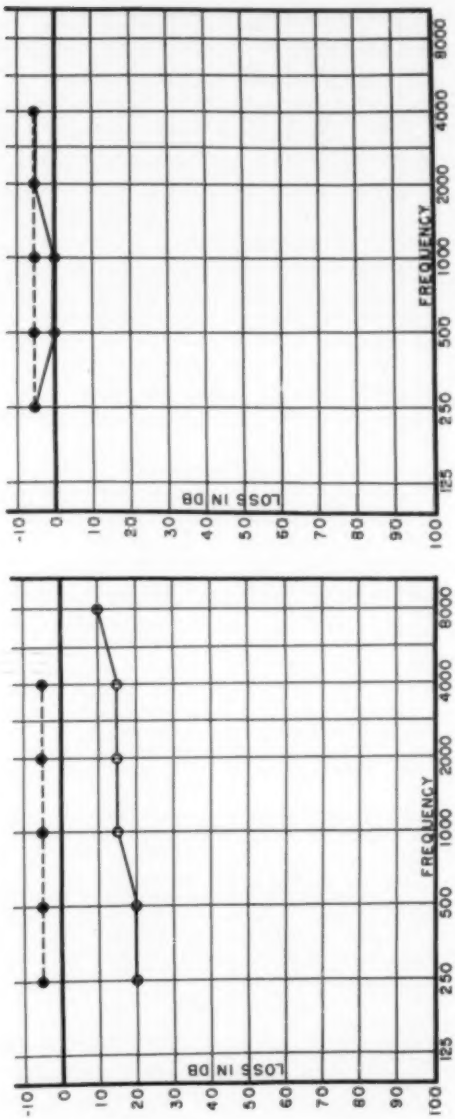
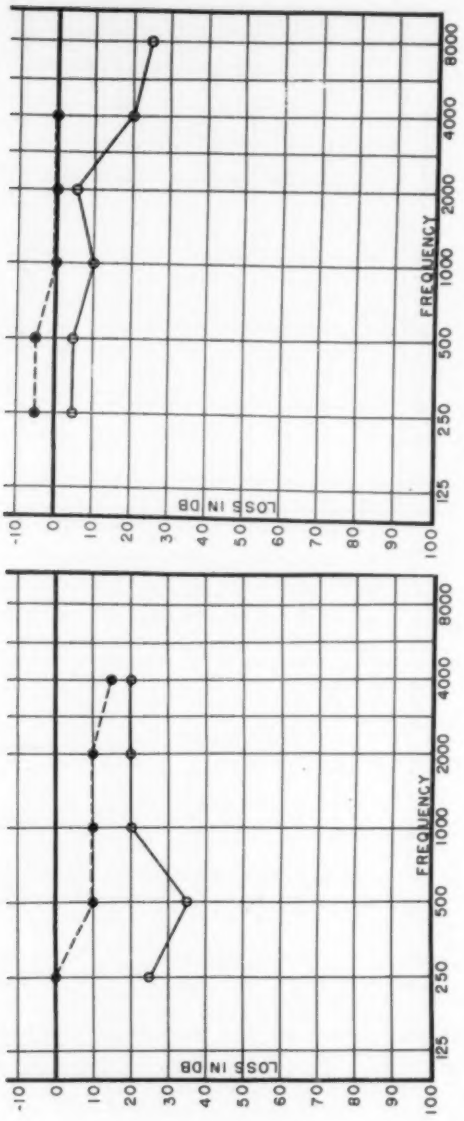


FIG. 3. A.—Age 5, 1945; B.—Age 17, 1957.



B.

A.

FIG. 4. A.—Age 5, 1946; B.—Age 16, 1957. Spontaneous hearing improvement.

GROUP NO. 2.

This group consisted of children with mild conductive losses of 15 to 35 dbs. in the three speech frequencies, who did not respond satisfactorily to therapy in childhood, but later regained their hearing after adolescence. This gain was apparently due to spontaneous regression of tubal lymphoid tissue.

CASE REPORT.

E. K., age five, was first examined in 1946 with a history of repeated upper respiratory infections and recurrent attacks of bilateral acute otitis media with drainage. The parents had been aware of a hearing loss for several years.

Examination revealed enlarged adenoids and tonsils, and their removal was recommended. The parents did not desire to have surgery performed, and no therapy was carried out, either at that time or subsequently (see Figs. 4-A, 4-B).

GROUP NO. 3.

This group consisted of children with mild conductive losses that increased to loss of serviceable hearing in adulthood, apparently due to otosclerosis. Subsequent surgery now being performed on this group is proving the diagnosis of otosclerosis to be correct.

CASE REPORT.

R. K., age 15, was first examined in 1954. She had noted a bilateral hearing loss for six months which was discovered accidentally, and which she stated was not progressive. She complained of some tinnitus, and stated that she heard better in noisy places. There was a positive family history of otosclerosis.

Examination revealed essentially normal otologic findings. The tonsils and adenoids had been removed at an earlier age, and the nasopharynx was free of lymphoid tissue.

Two years later another hearing examination was carried out, and the hearing loss had progressed considerably during adolescence. A mobilization operation was performed at that time with restoration of serviceable hearing, as per the following audiograms. The findings at surgery were characteristic of otosclerotic fixation (see Figs. 5-A, 5-B).

GROUP NO. 4.

This group consisted of children with a marked conductive loss in childhood, of 45 to 55 dbs., who showed the same degree of loss in adulthood ten or more years later, apparently

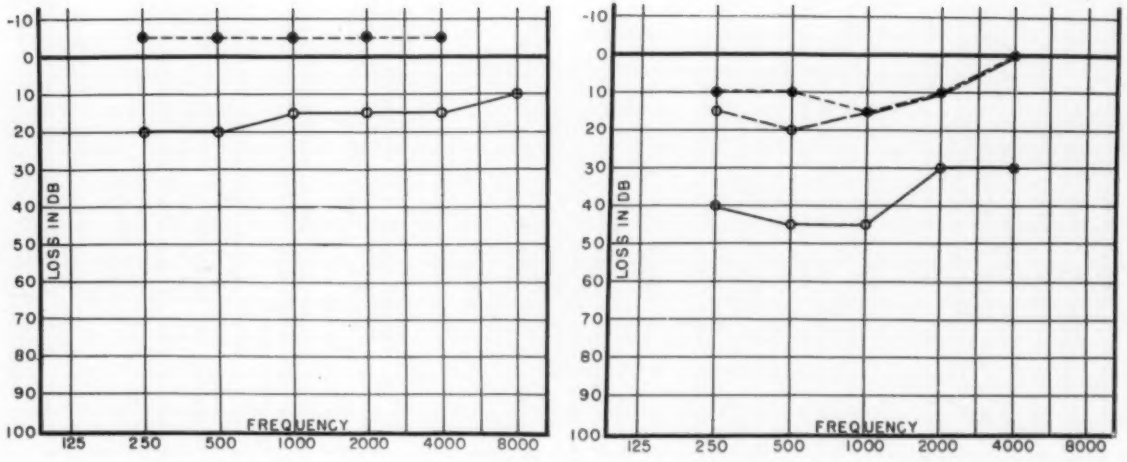
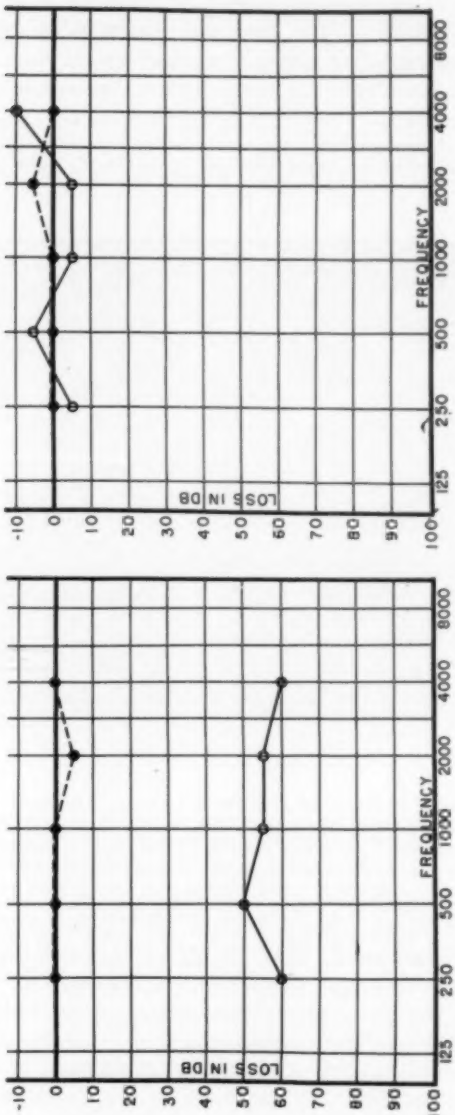


Fig. 5. A.—Age 15, 1954; B.—Age 18, 1957. Note further preoperative loss of hearing to a 40 db. average improved following mobilization to a 15 db. average. Circle-continuous line, preoperative; circle-line, postoperative air conduction; solid dots with dashes, bone conduction curve.



A. B.

Fig. 6. Age 10, 1945; B.—Age 22, 1957. Closure of the bone-air gap following mobilization surgery.

due to congenital footplate fixation. Subsequent surgery now being performed on this group is proving the diagnosis of congenital footplate fixation to be correct.

CASE REPORT.

V. M., age ten years, was first seen in 1945. The parents stated the hearing impairment was first noted at three years of age, and that the child was rather slow in learning to talk. The parents did not notice

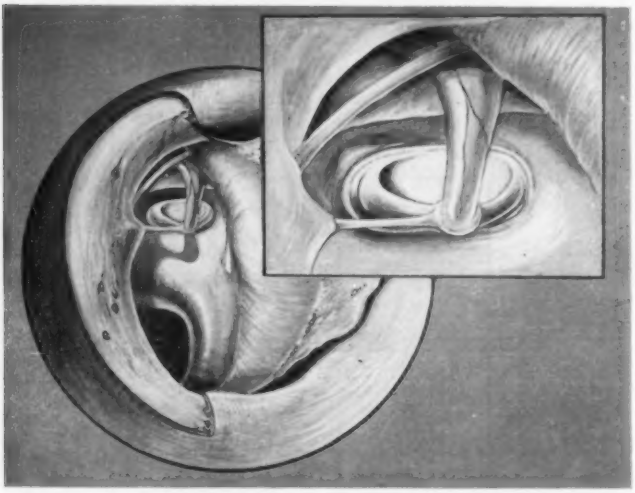


Fig. 7. Normal Footplate.

any progression in the loss and there was no history of ear disease. The child was unaware of tinnitus and the family history was negative.

Examination was essentially normal except for the audiometric findings, which revealed these characteristic audiograms (see Figs. 6-A, 6-B).

In a child under 15 years of age the diagnosis of otosclerosis is difficult, because the key factor of progression cannot be definitely established. The degree of conductive loss at the time of discovery may vary from an average of 15 dbs. to 35 dbs. in the three speech frequencies, depending upon the degree of otoscelerotic footplate fixation. During adolescence progression of hearing loss due to otosclerosis is frequently accelerated rapidly.

If a child has normal physical findings, has a conductive loss not to exceed 35 dbs. in the three speech frequencies and does not respond to routine therapy, the diagnosis may be clinical otosclerosis. Confirmation of this tentative diagnosis can be achieved only by noting further progression of the hearing loss over a period of time, or by surgical exploration and examination of the stapes footplate.

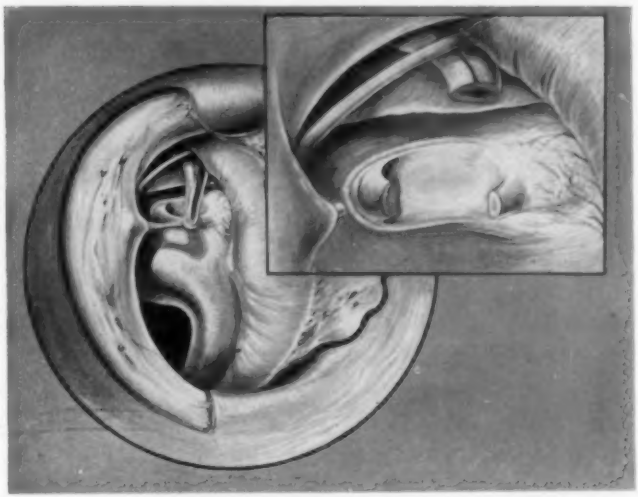


Fig. 8. Otosclerotic Footplate.

The diagnosis of congenital footplate fixation in the child may also be difficult, due to the lack of a history of progression of the hearing impairment; therefore, the diagnosis of congenital footplate fixation in children is based on normal physical findings and the characteristic flat 50 db. pure conductive loss on the audiogram.

In view of our long term studies of conductive impairment in children, it seems logical that any child under 15 years of age with a purely conductive hearing impairment approximating 50 dbs. has congenital footplate fixation rather than

clinical otosclerosis. An analysis of our operated cases of congenital footplate fixation in children confirms this hypothesis.

OBSERVATIONS AT SURGERY.

Surgical exploration by the stapes mobilization approach reveals rather characteristic findings in cases of congenital footplate fixation (see Fig. 7).

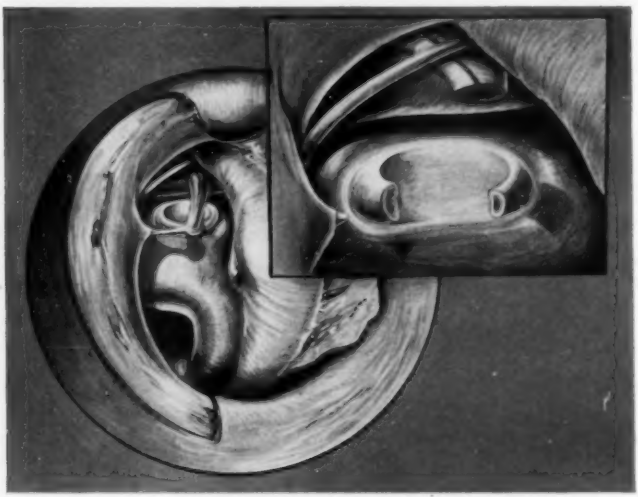


Fig. 9. Congenital Footplate Fixation.

The normal stapes footplate has a characteristic appearance. There is no increase in vascularity, and the margins of the footplate and the annular ligament are distinct. The central portion of the footplate is thin and usually has a bluish appearance. Gentle palpation reveals uniform mobility (see Fig. 8).

The otosclerotic footplate has an increased vascularity, usually in the anterior area. The margins of the footplate and the annular ligament are difficult to visualize anteriorly,

but usually are readily visualized posteriorly. The central footplate area is often thin with a bluish discoloration. Otosclerotic bone frequently engulfs the anterior crus at its footplate attachment. Whitish otosclerotic plaques are often seen spreading over the surrounding bone of the otic capsule. Palpation reveals fixation of the footplate, more often in the anterior region (see Fig. 9).

In congenital footplate fixation there is no increase in vascularity. The central portion of the footplate is not thin,

TABLE I.

A Preliminary Report of Twenty-Three Operated Cases of Congenital Footplate Fixation.

Age of Discovery of Hearing Loss.		
Average age	10 years	
Youngest age	2 years	
Oldest age	33 years	
(87% noted hearing loss before 14 years of age).		
Age at the Time of Surgery.		
Average age	33 years	
Youngest age	6 years	
Oldest age	60 years	
(Five cases were over 50 years of age).		
Preoperative Air Conduction.		
Average loss	50 dbs.	Average in the three speech frequencies
Minimum loss	40 dbs.	
Maximum loss	65 dbs.	

and the bluish discoloration is usually not present. The margins of the footplate and the annular ligament are difficult to visualize, since the footplate bone blends into the bone of the surrounding otic capsule. This diffuse uniform bony fusion may be thin or thick. If thin, the footplate can be readily mobilized; if thick, mobilization cannot be attained by manual pressures. Gentle palpation reveals uniform and total fixation of the entire footplate.

SURGICAL RESULTS.

All twenty-three cases were explored by the stapes mobilization approach. Mobilization of the stapes footplate was attempted by needle and chisel techniques.

Twelve cases (52 per cent) had a significant gain of 11 decibels or more in the three speech frequencies. Eleven of these twelve cases reached the 30 decibel level or better three weeks following mobilization. Eleven cases (47 per cent) remained essentially unchanged (within 10 decibels of the preoperative level in the three speech frequencies). In five cases (22 per cent) the stapes footplate could not be mobilized by manual means. Of these five cases three were subsequently fenestrated and all three reached the serviceable level of 30 dbs. or better.

This series of cases of congenital footplate fixation is too small, and the time factor too short, to establish conclusions in regard to permanency. Cases operated successfully one year ago, however, seem to be maintaining their post-surgical hearing improvement.

CONCLUSIONS.

1. The diagnosis of congenital footplate fixation is established by observing the characteristic flat audiometric curve by air conduction approximating 50 dbs. in the three speech frequencies. The hearing loss in these cases is non-progressive.

2. Congenital footplate fixation is a definite entity which can be diagnosed clinically and confirmed at surgery.

3. Mobilization in cases of congenital footplate fixation is usually more difficult than in cases with otosclerotic fixation. If the footplate cannot be mobilized the patient is an ideal candidate for subsequent fenestration surgery.

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SYMPOSIUM.

STAPES MOBILIZATION TWO YEARS LATER.

MODERATOR:

H. P. HOUSE, M.D., Los Angeles, Calif.

PANEL:

MERLE LAWRENCE, M.D., Ann Arbor, Mich.
J. R. LINDSAY, M.D., Chicago, Ill.
VICTOR GOODHILL, M.D., Los Angeles, Calif.
GEORGE E. SHAMBAUGH, M.D., Chicago, Ill.
C. M. KOS, M.D., Iowa City, Ia.
E. P. FOWLER, JR., M.D., New York, N. Y.
P. E. MELTZER, M.D., Boston, Mass.

CLOSING REMARKS:

SAMUEL ROSEN, M.D., New York, N. Y.
HOWARD P. HOUSE, M.D., Los Angeles, Calif.

OFFICIAL DISCUSSIONS

R. J. BELLUCCI, M.D., New York, N. Y.
F. A. SOOY, M.D., San Francisco, Calif.
S. H. BARON, M.D., San Francisco, Calif.
H. F. SCHUKNECHT, M.D., Detroit, Mich.
F. R. GUILFORD, M.D., Houston, Tex.
JOSEPH SULLIVAN, M.D., Toronto, Canada
THEO. E. WALSH, M.D., St. Louis, Mo.

OPEN DISCUSSION:

MOSES LURIE, M.D., Boston, Mass.
JUERGEN TONNDORF, M.D., Iowa City, Ia.
KENNETH M. DAY, M.D., Pittsburgh, Pa.
GORDON HOOPLE, M.D., Syracuse, N. Y.
LEE SHAHINIAN, M.D., Redwood City, Calif.
HOWARD P. HOUSE, M.D., Los Angeles, Calif.

CLOSING DISCUSSION:

MERLE LAWRENCE, M.D., Ann Arbor, Mich.
VICTOR GOODHILL, M.D., Los Angeles, Calif.
GEORGE E. SHAMBAUGH, M.D., Chicago, Ill.
C. M. KOS, M.D., Iowa City, Ia.
E. P. FOWLER, M.D., New York, N. Y.
PHILIP E. MELTZER, M.D., Boston, Mass.
SAMUEL ROSEN, M.D., New York, N. Y.
HOWARD P. HOUSE, M.D., Los Angeles, Calif.

MODERATOR HOWARD P. HOUSE, M.D.: Two years ago in Montreal, this panel analyzed its combined knowledge and efforts regarding stapes mobilization. Today this same panel will bring to you newer thoughts and concepts in our thinking regarding stapes surgery during the past two years.

In the interests of time conservation your Moderator today will ask each member one or two critical questions. This will allow more time for audience participation and discussion at the conclusion of the panel presentation.

First, I would like to call on the new member of our panel—Dr. Merle Lawrence, will you discuss the function of the middle ear as related to stapes mobilization surgery?

MERLE LAWRENCE, Ph.D., (Ann Arbor, Mich.): In order to speed the discussion along I shall use the blackboard to draw a schematic diagram of the ear, so that we can see what happens when we change parts of it. We have the malleus (drawing on board), incus, stapes and inner ear, which I draw diagrammatically to show the endolymphatic system surrounded by the perilymphatic. This gives us a picture of the normal ossicular chain, the drum membrane, malleus, incus, stapes and the scala media.

The first essential for the production of hearing is the presence of the sensory cells; they must be present in the scala media and available for vibration. The second item of importance is the relay of vibratory energy to these cells. The primary purpose of the ossicular chain is to provide a match between the air and inner-ear liquids so the vibration can get into the fluids surrounding the sensory cells and

cause them to vibrate. The round window membrane acts as a relief point so the in-and-out vibration of the ossicles can be transmitted through the scala to the round window, which then bulges in and out.

There are several things that can happen to prevent this process: first, the ossicular chain can be disrupted through the removal of the incus or the disarticulation of the incudo-stapedial joint. A measurement of hearing in this case, an experiment which has been done many times on animals, shows that such a disarticulation gives a loss of around 60 db. The reason for this is that we now have a useless drum membrane that is impeding the sound vibration from getting into the inner ear; also, the two windows are in the path of the vibrations that do get through. Sound hitting the two windows in phase and with equal intensity will be cancelled out at the sensory cells which lie in the internal path between the windows.

If the drum membrane is removed, as has been done both clinically and experimentally, the hearing will improve by about 15 db. This leaves a loss of around 45 db., part of which can be accounted for by the cancellation effect of the sound hitting the two windows. If, by some means, it were possible to get sound to one window alone, thus preventing the cancelling effect, hearing could be brought up to a 25 to 30 db. level. This, of course, is quite important because this is in the range of practical hearing—without an ossicular chain.

Now let us suppose there is a stapes fixation. This may produce a loss of 60 db. or more. Disarticulating the ossicular chain in this case will not produce more loss because the stapes is immobile and useless. Although there is sound going in only one window the fluid can not respond to the vibration because it is blocked off at the other end by the fixed footplate of the stapes.

In this case the first thing to try is, of course, breaking these adhesions to re-establish a normally moving chain. If successful this is ideal, because the normal situation is restored; but, if this cannot be done, there are several pro-

cedures that can be carried out which, if successful, can return hearing to the 45 db. level.

One of these procedures is to pull or break the stapes out of the oval window. I would like to present two slides which show the results of such an operation on an experimental animal. It is remarkable that the stapes can be pulled out without greatly changing the cochlear response from that produced with a mobile stapes with no ossicular chain.

I would like to emphasize one thing at this point. I am not advocating surgical procedures; I am talking about the mechanical properties of the ear under various conditions of surgical rearrangement in experimental conditions.

In the first slide is shown a running-time record of the electrical response from a cat's cochlea when presented with a continuous 2000 c.p.s. tone. Such recordings are made possible by an electronic system which I shall not describe. At the beginning of the record the stapes is fixed. This is accomplished in the anesthetized cat by inserting a needle deeply into the cartilagenous portion of the Eustachian tube, a procedure which directs the remainder of the needle in a posterior direction directly past the stapes, wedging it against the promontory.

The incus is removed, a procedure which, as long as the stapes is fixed, produces no change in the response. Mobilization of the stapes by removing the needle, under these conditions, gives an improvement of about 12 to 13 db.

As seen in the slide the next step is to pull out the stapes completely and, momentarily there is no change in response from that of a mobile stapes. Such operations were separately reported as a surgical procedure on humans in 1892 by Blake and Jack; however, they soon abandoned it.

Our next step, referring again to the slide, was to investigate the nature of sound transmission under the above conditions. First, the now-open oval window was plugged solidly by pushing wax down into the niche. There is an obvious drop in the response. The response returns upon removal of the plug. Plugging the round window in the same way

again produces a drop in the response. Removal of this plug brings the response back to the both-windows-open condition. Obviously, in the presence of a normal inner ear the worst condition, aside from having both windows blocked, is to have one window closed and the other opened. In the presence of a fixed stapes the situation can be improved by mobilization or temporarily by the drastic procedure of pulling the stapes out of the oval window.

In the above experimental procedures the incus had been removed, so the drum membrane and its attached malleus were useless, having no direct connection with the mobile stapes or open window. By removing this barrier to the sound, the response improves even further.

The second slide shows the audiometric results for several frequencies. The one curve shows the loss produced by the fixed stapes and the second shows the loss, a considerable improvement over the original condition, of mobilizing the stapes (and removing it) plus elimination of the drum membrane, malleus and incus. It shows that through these procedures a marked improvement in hearing can be produced temporarily in the laboratory. It does not indicate what the permanent results would be or what surgical complications would be encountered in the human.

Despite the demonstrated improvement the average theoretical results would still show a hearing loss of about 45 db. This is not enough of an improvement. To be practical 15 more db. must somehow be restored. There is another mechanical property which can be considered in attaining this additional 15 db.

We have mentioned that this 15 db. loss is produced by sound hitting both windows in phase and with equal intensity. This can be prevented by creating a situation that leaves one window open to the sound and protects the other without blocking it; that is, leaving it free to act as a relief point for the in-and-out motions of the other window.

There are two ways by which such a situation can be accomplished: one is by means of a skin flap over the open oval window or over a fenestra in the horizontal canal (as in the

Lempert operation) which then is so attached that an air space is left around the round window but at the same time acting as a barrier between the sound and this window. Any opening in this barrier will, of course, not bring about the desired improvement.

Another procedure is theoretically possible in the case of a firmly anchored stapes. This consists of opening up the middle ear cavity by removing the drum membrane, malleus, and incus, which are useless in the face of an immobile stapes, and making a *minute* hole into the vestibule of the inner ear. This hole must be small, because it has to offer acoustic resistance to the entering sound. Such resistance cuts down the intensity of sound entering on the vestibular side and thus reduces the cancelling effect. Any larger hole, such as was produced in the experiment shown on the slide by pulling out the stapes, offers no resistance at all to the incoming vibrations. The tiny hole reduces the inner-ear cancelling effect and acts as a mechanical relief point for the unimpeded sound entering through the round window; so it is possible to obtain, by this method, an improvement up to the average theoretical level of 25-30 db. as in the fenestration operation, and depending upon the natural acuity of the operated ear the results may be even better in some cases.

I would stress that aside from the mechanical considerations mentioned here there are many physiological and medical problems involved. Making a small hole in this manner may be fraught with dangers because it involves sticking into the vestibule a sharp instrument which could puncture the utricle and so injure permanently the endolymphatic system; also, the hearing results following such a pin-point perforation would more than likely fail to be permanent.

If the initial attempts to restore a normally-operating ossicular chain through mobilization of the ossicular chain fail, it appears, when all factors are considered, that one should, for the patient's sake, resort to the now-proven fenestration operation despite the various other mechanical possibilities.

Something should be learned from past experience, but

we see an almost exact duplication and progression of techniques tried and abandoned years ago. At first, in the final decade of the last century, there was mobilization which was followed by stapes extirpation and holes drilled in various regions. Holes were drilled through the stapes, through the promontory, into the posterior canal and finally the horizontal canal. I predict that in about 1980 somebody is going to rediscover fenestration of the horizontal canal.

MODERATOR HOUSE: Dr. Lindsay, would you please discuss the pathology of extensive otosclerosis that might require footplate techniques for successful stapes mobilization?

JOHN R. LINDSAY, M.D. (Chicago, Ill.): The histopathology is the one factor of stapes ankylosis which does not change, but as years go on we are more aware of the many variations which exist. I thought it would be most informative to show a few of the really extensive cases of otosclerosis which would need some procedure directed at the footplate in order to be mobilized successfully.

I am indebted to Dr. Hilding of Duluth and to Dr. Keeler of Oakland for the four specimens that will be shown.

The first two slides are from Dr. Hilding's case. The first slide shows four sections through the same stapes to show how the condition varies. Near the upper border the footplate is free at one side but extensively involved in otosclerosis and fixed at the other edge. The section further down shows the fixation and greater extension of the otosclerosis. Further down it extends well over the footplate, but still the posterior border is free in its upper part. Lower down there is still a part of the footplate free where it is connected with the posterior crus. The anterior crus is hopelessly involved in the otosclerotic mass. This, as you see, suggests that the procedure first suggested by Fowler might be adaptable, that is, an anterior crurotomy and mobilization of the remainder of the footplate by creation of a fracture, leaving a mobile part of the footplate connected through the posterior crus to the remainder of the chain.

The second slide shows a more difficult problem. The situation is essentially the same in that part of the footplate is

free, and one border is free from fixation but the anterior crus is hopelessly involved in the otosclerosis and the posterior crus is also slightly involved. At one point it is touching on the otosclerotic mass and is slightly fixed. Here the problem would be to get this free part of the footplate mobilized and also to get the posterior crus free without causing a fracture where it is involved by the otosclerosis. It is questionable whether this could be done successfully.

The third slide, from Dr. Keeler's case shows a different situation. This footplate is completely fixed all the way around the circumference. The otosclerotic mass involves the footplate so that to mobilize this footplate it would be necessary to go through fairly active areas of otosclerosis. This would require some method of mobilizing the footplate, whether by chisels or by whatever method the operator might prefer.

The next slide from the opposite ear of Dr. Keeler's case is somewhat similar. This is also completely fixed all around the circumference, and the otosclerosis extends into the footplate. The crura are not too bad, although one crus is extensively involved by otosclerosis. This would certainly require some method of attack on the footplate in order to have any chance of success. The method of attack might vary with the particular operator.

MODERATOR HOUSE: Dr. Victor Goodhill, will you discuss the selection of cases for mobilization? Have our thoughts regarding selection changed any in the last few years? Please discuss this problem from two standpoints: First, the indications and selection for the initial operation; second, the indication for revision of the initial operation.

VICTOR GOODHILL, M.D., (Los Angeles, Calif): These recommendations represent the composite views of the panel. There are occasional exceptions to all of these statements, depending upon the judgment and experience of the individual surgeon:

Minimal requirements for selection of candidates:

1. An air bone gap of at least 25 db. in the speech fre-

quencies and the demonstration of a negative Rinne with the 500 and 1000 cycle tuning forks.

2. A bone conduction threshold not lower than 40 db. in the speech frequencies.

3. A speech discrimination score with PB words not lower than 75 per cent.

4. Unilateral cases, if carefully confirmed by Weber lateralization, by quantitative masking and by Barany noise machine masking, providing that there is an expectation to reach the 30 db. level, thus giving that patient the possibility of restoration of binaural hearing.

5. It is pretty much our unanimous feeling that the poor ear should be done first.

6. In general we do not urge or recommend simultaneous bilateral surgery.

7. The choice of time for the second ear should be several months, about four months later at least.

These represent our feelings about minimal requirements for primary surgery.

Now as to revisions in general, chances for improvement are best with the first operation and become considerably less with successive reoperations.

The surgical observations of the surgeon at the time of the first operation are often crucial in a decision regarding revision. (From here on there appeared quite a diversity of opinion about revisions, and I am going to give them to you as we discussed them.)

a. Some on the panel felt that disruption of the ossicular continuity at the initial operation would be a contraindication to further surgery.

b. Others felt that such disruption would not contraindicate revision attempts by the use of newer techniques.

c. Some advise revision if there was no improvement at the first operation, if the surgeon feels that a newer tech-

nique in his hands offers hope for mobilization, which did not exist at the first operation.

d. Some advise revision in any case if an initial improvement regresses at any time. Others feel that such revision should be done only if the initial gain persisted at least nine months. Some felt that revision should be done only once; others, many times. In conclusion there was no true unanimity of opinion on all of these aspects of revision.

MODERATOR HOUSE: Thank you, Dr. Goodhill. I believe we conclude from this that we all agree on the indications for the initial procedure, but again you can see apparently there is no unanimity of thought pertaining to true indication for revision or on how many times the revision should be carried out.

Dr. Shambaugh is going to answer two questions that I will pose to him: First, what major technique changes have occurred during the past two years in stapes mobilization surgery?

GEORGE E. SHAMBAUGH, M.D., (Chicago, Ill.): Three major developments in technique have occurred since we first assembled this panel. One is the utilization of audiometric testing during the operation in the operating room. This was employed by several members of the panel two years ago and is now employed by all members.

The second is the utilization routinely of the operating microscope to give a magnification of 10 to 16 times for visualization of the footplate. Two years ago the majority of the panel relied upon two to two and one-half times magnification, and today all members of the panel agree that the operating microscope is essential for this surgery regardless of technique.

The third development, which is largely dependent upon the increased magnification of the operating microscope, is the direct attack upon the footplate. At the present time approximately half of this panel start with the transcrural attempt at mobilization, either on the incus, on the neck or on the head of the stapes and then proceed to the footplate, in

the event that mobilization attempts transcrurally are not successful and before fracture of the crura has occurred. The other half of the panel proceeds directly to the footplate using picks, chisels or other instruments applied to the margins to loosen it and obtain mobility. All members of the panel agree that in working on the footplate any perforation that opens into the vestibule definitely increases the danger to the labyrinth. Deliberate anterior crurotomy, as described by Fowler, is utilized in occasional cases by members of the panel, but in the majority of cases an attempt is made to mobilize the entire footplate.

In concluding on the technique of this operation I would like to make a little statement, which was suggested to me by Jim McLaurin, that there are two operations for otosclerosis.

MODERATOR HOUSE: May I interject just a moment before you get to this second part, by asking you your second question?

DR. SHAMBAUGH: Yes, indeed.

MODERATOR HOUSE: What are the thoughts of the panel on fenestration of the footplate as described by Dr. Rosen?

DR. SHAMBAUGH: At this time the majority of the panel members believe that a deliberate fenestra in the footplate without communicated mobility from the tympanic membrane to the mobilized footplate, or fragment of footplate, in all probability will not produce a lasting satisfactory level of hearing improvement.

Now may I make that last statement, that there are two operations available for otosclerosis? One is the fenestration operation and the other is the "frustration" operation, frustrating because of the unpredictability of its results.

MODERATOR HOUSE: Thank you, Dr. Shambaugh. Now I will call on Dr. Kos. (There is another place you might use the word "frustration." That has to do with attempting to arrive at statistical evaluation of results in mobilization surgery.)

I will ask Dr. Kos to describe to you some of these complexities of analysis, and then he will present to you our results during the past two years.

Dr. Kos, do these results look as though we are gaining or losing in our attempt to restore hearing through mobilization surgery?

C. M. Kos, M.D. (Iowa City, Iowa): I take this opportunity to thank each of the members of this symposium for cooperating so generously in providing me with these data. Since each has his own method of keeping records it was not easy for him to extract the precise information requested, but all of them made a valiant effort to comply. Some irritations were aroused. One began every reply to my inquiries with "Damn all questionnaires." Another when he reached the limit of his patience suggested sarcastically that if necessary he would write my paper for me. I wanted to accept his invitation. Others stuck to the point in spite of having some reason to rebel and delivered without protest.

I must give to Dr. Jim Shapley, our Director of Audiology, and to Mrs. Iles, his able assistant, the entire credit for handling the information received. Had I followed their original recommendations for the questionnaire, the entire job would have been easier; but my efforts to simplify a complicated task only compounded the difficulties, so Dr. Shapley and Mrs. Iles did the best they could to straighten things out. We have lots of figures, but to give them in detail would require more explanatory notes than there is time and space available; therefore a contraction will be presented.

The ten otologists on this panel, whose experiences range from 105 to more than 2,000 stapes operations, have produced an equally varied spread of results. Together this group has performed 7,420 surgical procedures to mobilize the stapes as of January 1, 1958. Two years ago the panel reported on 1,091 operations. Our total successes, of at least two months' duration, range from 25 per cent to 74 per cent, depending on each individual's criterion for success. Our average success according to these various standards is 49 per cent. When success is precisely defined as an average pure tone threshold

of 30 db. or better in the three essential speech frequencies the spread between us is reduced somewhat—the range being 25 per cent to 65 per cent. The average of the group in this category is 42 per cent. Two years ago this figure was 35 per cent. Four of the participants achieved 42 per cent or better. All of the rest, but one, stand very close by. Two of the panelists hit this target with a closure of the air-bone gap in every one of their cases. The others ranged from 13 per cent to 70 per cent. The highest figure in this category two years ago was 59 per cent.

It is notable that those who have performed the most operations have in general presented the more modest statistics. It may be that these panelists are stuck with less desirable statistics because they led the way through the maze of uncertainties, and now having passed on their experiences have saved the others from falling into the inevitable traps of exploration. I know that some of us have been experimenting in many ways with surgical techniques, and this is not always without its indebtedness to failure.

Regression of hearing following a successful surgical result is always an unpredictable barrier to permanent improvement. There are at least 12 recorded concepts of regression from this group. There may be more. It is difficult to reconcile the wide spread among us of the incidence of regressions. These range from 4 per cent to 32 per cent. The average is 15 per cent; however, when one considers that some of these participants are following patients whose operations took place three to five years ago and others are following those whose operations are of less duration, one possible explanation may be derived. Time takes its toll of regressions even though it be in decreasing numbers.

Operations resulting in worse hearing range from 0 per cent to 8 per cent in this group, depending on at least seven definitions of what constitutes "hearing made worse." The average is slightly over 3 per cent (about the same as it was two years ago). This percentage in 7,420 operations is probably acceptable if no other complications are involved; and according to the statistics of this group it is not so severe as it was once anticipated.

Our experiences of the past two years indicate that significant progress has been made in stapes mobilization. We are now aware that regressions continue to rob us of some of this progress and these are likely to become our prime target for investigation in the years to come.

The experiences acquired during the hectic period of assembling these data point out the very important requirement for developing and establishing a common language to bridge the conceptual differences between us and others reporting in this field. Several of my colleagues have also made this plea. Fifteen different criteria for success, 12 concepts of regressions, and seven definitions of what is "hearing made worse" call for conferences designed and conducted to resolve these discrepancies.

MODERATOR HOUSE: Thank you, very much, Dr. Kos.

You have my sympathy and I am sure the sympathy of Dr. Goodhill, because the two of us, in Los Angeles, got together before the Montreal meeting and attempted to go through this maze of information in an attempt to determine the results at that time, and you can see we were successful in convincing Dr. Kos that he should fulfill his term of duty on this occasion.

Now I will call on Dr. Fowler; are any significant complications very frequently found in stapes mobilization surgery?

EDMUND P. FOWLER, JR., M.D. (New York, N. Y.): I am sorry to report that this panel is no better sending statistical material to *me* than they were with Dr. Kos; as a matter of fact, I think they were worse. Perhaps I did not work as hard at it as Dr. Kos did, but there was no way in which I could put figures together, that would give you any idea of the complications of mobilization surgery in a statistical fashion. I believe it is most important to emphasize that the panel had about 3 per cent of cases that were made worse, as just reported by Dr. Kos.

You also notice that he mentioned that this ran between 0 and 8 per cent. He did not point out that of the 3 per cent only a half of one per cent were severely worse. By "severely

worse" we have agreed we mean more than 20 db. worse loss than the patients had before the operation. Throughout the panel there were only three dead labyrinths, and one of those occurred about a month after operation, suddenly and inexplicably. There is a question whether this dead labyrinth had anything whatsoever to do with the operation. There was 1 per cent of persistent perforations. There was a good deal of question in the minds of the panel whether this should be listed as a serious complication.

There was no facial paralysis that was permanent. There were three or four that developed a transient facial paralysis after the operation. They recovered, and this was annoying but not serious.

There was annoying persistent vertigo in three-tenths of one per cent. There was a great deal of question about the chorda tympani in the letters written to me. I could summarize the ideas of the panel as follows: In general it was felt that stretching the chorda tympani hard was worse than cutting the chorda tympani. Some of the panel felt that any injury to the chorda tympani was a serious matter, and all thought it should be avoided if possible.

I have just discussed with you the complications that were suffered by those on this panel. Throughout the country the story is quite different. There are several cases of permanent facial paralysis. There are several dead labyrinths. There are a few very annoying chronic otitis medias. One case developed mastoiditis, which could not be controlled and which eventually developed a base of the skull syndrome. He is still alive but has a paralysis of the nerves that go through the jugular foramen, the VIth nerve and I think the Vth. At any rate he had an extradural abscess at the base of the skull, and the patient is alive, I am sure, only because of hard work and chemotherapy.

Finally, I would like to mention that there have been *two deaths,* one from staphylococcus labyrinthitis followed by meningitis, which was not controlled with the most heroic therapy and operation. The other—this case thought to be a hospital staphylococcus that was resistant to all chemo-

therapy—is even more disturbing because of pneumococcus meningitis. This patient seemed perfectly all right for 10-15 days, except for a little suppuration, which is not too uncommon in the early cases, (even for this panel) but suddenly a few days after his last visit to his otologist he developed a coma with a pneumococcus meningitis which was not controlled by chemotherapy and he died.

MODERATOR HOUSE: Now I should like to call on Dr. Philip Meltzer, who will give us some of his views pertaining to the past, the present and the future of stapes mobilization surgery.

PHILIP E. MELTZER, M.D. (Boston, Mass.): It is my intention to offer a word of warning that the procedure of stapes mobilization should be undertaken with a complete awareness of its pitfalls.

This operation has fired the imagination of otologists throughout the world, and aroused in the mind of the layman a reaction brimming with hope for the alleviation of deafness. So many procedures for stapes mobilization have been devised that the time has come for a discussion of their rationale. It has never been denied that the restoration of the ossicular chain as a mobile unit for sound transmission is not valid; therefore, this operation stands on firm ground. Whether or not a stapes once liberated will continue to remain so, and which of the many procedures will accomplish this most often and permanently, are questions to be discussed at symposia of this kind.

I have observed the technique of some of the most practiced surgeons of this country, and my critical evaluation of this operation and my queries for the future are based on what I have witnessed, and my own experience.

In fenestration surgery we are confronted with several problems. Serous labyrinthitis and regeneration of bone in the fenestra are chief causes of failure. Surgical access to the critical area for making the fenestra was not too difficult, and, as you are well aware, the continued advance in technique practically insures the success of this operation in well selected cases; but what can we look forward to, anatomically and surgically speaking, in the region of the stapes—a region

so confined, in which instrumentation is so extremely limited, in which the structures must remain *in situ*, in which the surgical efforts involve a diseased area, and in which nothing is removed, so that the diseased focus remains? When bone, whether otosclerotic or not, is broken or cut through, I would assume that Nature's reparative process would sooner or later take place, and it does in many cases. On the other hand, the fact remains that repair does not *always* occur, and thereby seems to refute my negative assumptions. Since my topic is concerned with the prospects of stapes surgery, I must at this point declare that just as the problems of fenestration surgery were solved, these stapes problems will also be solved.

A notable observation has been that many patients have reached a level of hearing to a degree that is attained less often by fenestration. The joy of both doctor and patient following a brilliant result with stapes mobilization is not infrequently tempered by the disillusionment of ultimate failure, or if not in failure, in at least a recession of the hearing from the high degree of initial improvement. The recession may occur not only in several weeks but as late as 20 months afterward.

To my recollection, never in the history of otology has there been the development of so many techniques as in stapes mobilization. The early enthusiasm for this operation stimulated the dormant inventiveness of many otologists throughout the world, and the most intricately designed instruments have appeared and disappeared. For the present I do not favor mobilizing the stapes by mechanical means. I believe, as Rosen does, that the mobilization is best accomplished by the sensitiveness of touch and the control of pressure in the surgeon's fingertips, and for this reason, in my opinion, no mechanical instrument, at present, can be substituted.

The prospects of stapes mobilization are threatened when any otologist declares that a *promise* may be made to the patient that the hearing *will not* be worsened, and that if the attempt to mobilize the stapes should fail, the otologist *can always* resort to the fenestration procedure. This statement would be true more often if the otologist knew *when* to stop

in his desire to free the stapes, and ceased to pursue traumatizing techniques that may preclude the fenestration operation.

I now want to discuss the delicate and controversial issue of the otologist's qualifications for the performance of this type of surgery. The stapes itself is so variable and fragile in structure; so loosely united with the incus, and so deeply placed in the fossa in such close relationship with the facial nerve, that this area is particularly vulnerable to accidental injuries and fracture, in the hands of even the most competent surgeons. Just as the casualties of bad fenestration surgery have been observed over the years, we are now observing the casualties of bad stapes surgery. The surgeon who is trained, but inexperienced, should not even consider attempting this procedure without first observing and learning in the most painstaking manner. He must also be trained in clinical audiology as well as in all phases of temporal bone surgery, so he will be equipped to render a complete service to his patient.

It is easy for me to stand before you and make these stipulations, but where is a man to get this kind of training? That is a problem that those persons in charge of residency training programs must solve by providing the training that will insure competence. Perhaps another solution will be that the more experienced otologists will act as preceptors in their private practices, or that teaching institutions will provide courses for qualified men.

Each one of us should examine his conscience to determine whether we are honestly equipped to perform the delicate maneuvers mandatory for the procedure of stapes mobilization. Then and then only can we give to the patient the type of care that is so rightfully expected of us.

MODERATOR HOUSE: Now I will call on Dr. Sam Rosen to tell us of any comments that he would like to make regarding this work. Further, I would like to ask Dr. Rosen this one question, how does it feel to have stimulated all of this interest in otosclerotic surgery?

SAMUEL ROSEN, M.D., (New York, N. Y.) (Closing): I don't know quite how to answer Howard House's question as

to how it feels to have started all this stapes surgery. On April 2, 1952, while I was testing a patient to see whether his stapes was rigid enough to have the Lempert operation, I mobilized the rigid footplate of the stapes by pressure on the neck and the hearing improved immediately. That patient still has normal hearing. I think that had I or anyone else, like Rip Van Winkle, gone to sleep on April 3, 1952, and awakened today to hear this panel and see the beautiful demonstration of middle ear structures by Dr. Hough, he would feel that a very pleasant dream had indeed come true.

It is very impressive to hear Dr. Merle Lawrence give us some understanding of the mechanism by which hearing is restored by my fenestra ovalis procedure.

I would like to show one slide to demonstrate whether it is possible to have lasting improvement following fenestration of the oval window. Of 141 successful cases, eight or 5.7 per cent, have regressed partially; 5 per cent have regressed completely and the number that have maintained the hearing improvement is 89.3 per cent of these 141 cases. We have data on 72 of these successful cases for periods of three months to a year; on 57 cases for periods of one to two years and in 12 cases the hearing has been maintained for periods of two to three years.

I should like to say to this entire Society that I was wrong when I said that this was a simple operation. I would like to erase that from the minds of all of you. It is not simple, and as time goes on it becomes more exacting.

MODERATOR HOUSE: We will now call upon our official discussors to give us three or four minutes of discussion of their experience, whether they agree or disagree with those on the panel, or give any newer thoughts they might have to offer at this time in their opinions of mobilization surgery during the past two years. First, I will call on Dr. Bellucci of New York.

RICHARD J. BELLUCCI, M.D. (New York, N. Y.): I am sure the official discussers are privileged on this occasion as they are not limited to discuss only Dr. House's one question.

I have two or three points I would like to make: first, in our experience, in order to obtain a consistently satisfactory improvement in

hearing the continuity of the ossicular chain must be maintained; therefore, techniques which depend upon the strength of the crura for mobilization have been discarded, and all forces are applied directly to the footplate at the point of maximal fixation. With the microscope and with direct visualization of the point of the needle it is possible to obtain visual as well as tactile control.

We have done considerable experimental work on animals; have fractured the footplate, have removed the stapes from the oval window and a variety of these operations. We have found that considerable tissue reaction occurs following trauma to the oval window, and we have experimentally induced stapes fixation by these traumatic procedures. We believe that mobilization of the stapes operation should be done with the least trauma to bone and mucosa. This operation should be a gentle procedure done carefully, and slowly, exploring the weaknesses in fixation which will permit mobilization by the simplest means. A very careful pre-mobilization evaluation of the pathology, therefore, is required before mobilization is attempted. By this pre-mobilization examination we are able to understand the pathology and to modify the technique of mobilization accordingly. In using the simplest means of mobilizing, trauma to the stapes and footplate is reduced.

If we can leave the joint and the stapes mobile and as normal as possible we believe we have achieved our goal. I believe that excessive trauma will certainly induce refixation.

I would like to show the classification we use as a guide for stapes mobilization.

(Slide). We have devised a classification based on the anatomical and pathological findings; the degree and position of the otosclerosis at the footplate. This is Class I; the characteristics of this Class I can be summarized as follows: Show no narrowing of the stapedial vestibular joint; the footplate is blue; palpation of the posterior end reveals motion, and palpation of the anterior end reveals that there is some degree of fixation; 36 per cent of the cases studied were found to have the characteristics of Class I. It is obvious here that any technique will mobilize this type of fixation; however, we placed the needle point right at the junction of the crus and the footplate. Slight pressure usually causes mobilization.

(Slide). Class II: the characteristics of Class II are narrowing of the stapedial vestibular joint but not involving the anterior crus, or then it may be the posterior crus. The footplate is blue; due to the flexibility of the stapes slight motion can be seen on palpation at one end of the footplate. In this slide at the anterior end there is definite fixation. Sometimes the footplate is involved as shown. In this group we find the most difficulty, as it requires the application of the mobilizing forces directly to the point of the otosclerotic fixation, having to go through the lesion and causing some degree of trauma. We therefore, have the highest rate of refixation in this type of case. There were 41 per cent of these cases in our study, which means that the largest group belongs to this Class II.

(Slide). Class III is advanced otosclerosis involving the anterior crus, the footplate and the vestibular wall. Only 14 per cent of the cases were found to be in this group. Mobilization in this group of cases is done by means of the anterior crurotomy of Fowler, where the lesion is circumvented. Part of the footplate and one crus operate together for the transmission of sound to the oval window. The anterior crus is divided above the otosclerotic lesion at this point.

(Slide). In Group IV we have the group of cases which are unfavorable

for the mobilization operation. Here is one where the facial nerve and the promontory have reduced the oval window niche to a slit, and the footplate is, of course, not visible. This case is anatomically unfavorable for mobilization operation.

(Slide). Where there is a large otosclerotic mass involving the whole footplate, mobilization is a difficult procedure, and I believe fenestration is the procedure of choice.

(Slide). Here where both crura are affixed by the otosclerotic lesion, even though the footplate is blue and does not show evidence of infiltration, the final result is usually not satisfactory, as the continuity of the ossicular chain cannot be preserved.

MODERATOR HOUSE: I would now like to call upon Dr. Frank Sooy of San Francisco.

FRANCIS A. SOOY, M.D. (San Francisco, Calif.): I would like to show you very briefly four types of cases that can, we feel, be confused in the pre-operative diagnosis of clinical otosclerosis.

Many otologists have been increasingly concerned with the number of patients seen in consultation, who present ordinary office puretone audiograms which are typical of otosclerosis but who, on further study or surgical exploration, are found to have some other clinical condition.

The excellent paper presented by Dr. Howard House at the American Otological Society, calling our attention to 26 cases of congenital anomalies of the stapes footplate simulating otosclerosis, is one such condition wherein confusion might arise, particularly in children. His finding on this syndrome in 1 per cent of his stapes mobilization cases is particularly significant, as is the fact that in most instances, this can be established clinically before surgery.

We feel that the time has now come to evaluate critically our conductive hearing losses in order to catagorize these patients into favorable cases and unfavorable cases, and we are now putting our first 500 cases on punch cards in which over 500 items are indexed in order to evaluate various pre-operative tests, in the hope that a more accurate prognosis can be made by means of more complete preoperative testing.

Four patients illustrating diagnostic problems are presented:

I. Congenital Stapes Anomalies:

This seven-year-old boy (see Fig. 1) was recognized as an anomaly preoperatively by the presence of hand deformities, and was successfully treated in the premobilization period by fenestration.

II. Unilateral Complete Hearing Loss with Inadequate Masking:

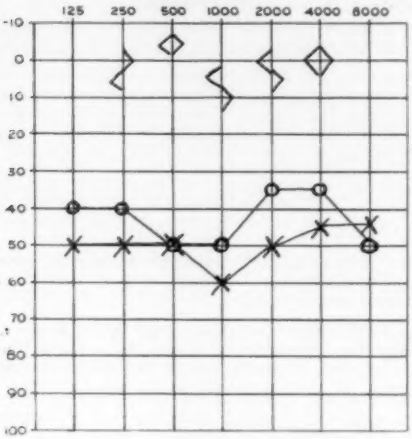
Fig. 2 shows the usual unmasked office audiogram in this condition, while Fig. 3 demonstrates the effect of masking at the usual maximum of 50 db. with the ordinary audiometer. Fig. 4 reveals the true audiogram, when masking is carried out at levels of 90 db. under research conditions using white noise, or sawtooth masking. Since many audiometers provide only 50 to 60 db. of masking this is a real problem.

III. Use of Psycholgalvanic Skin Reflex Audiometry (PGSR):

Fig. 5 shows the audiogram of a five-year-old girl who was referred for stapes surgery on the basis of PGSR testing only. Subsequent study by conventional methods using an audiologist trained in children's testing reveals a pure perceptive loss (see Fig. 6).

NAME J.R. AGE 7

PURETONE TEST DATE OF TEST 6-3-55



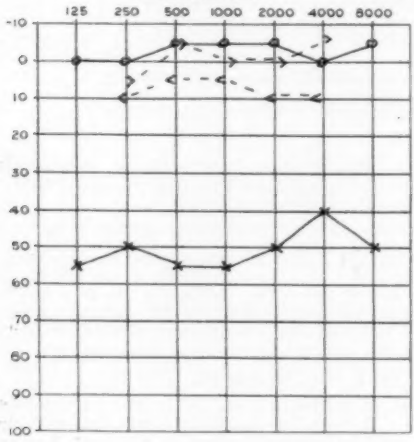
SPEECH TESTS

	L	R	FF
SRT			
ARTIC SCORE			
SAI			

Fig. 1.

NAME AGE

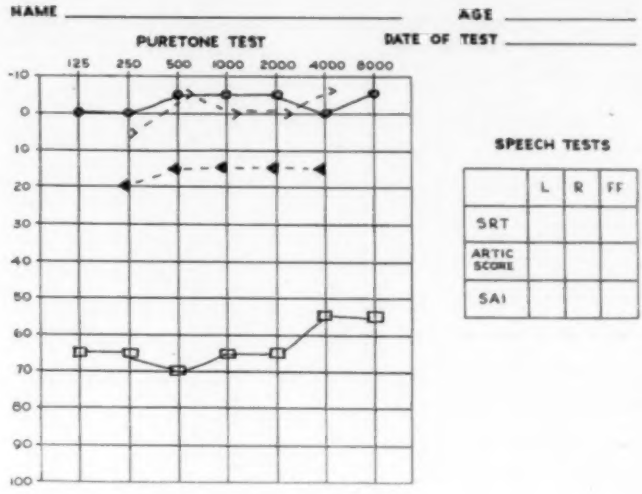
PURETONE TEST DATE OF TEST



SPEECH TESTS

	L	R	FF
SRT			
ARTIC SCORE			
SAI			

Fig. 2.


NAME
AGE
PURETONE TEST
DATE OF TEST
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SPEECH TESTS
L R FF
SRT
ARTIC SCORE
SAI

Fig. 3.

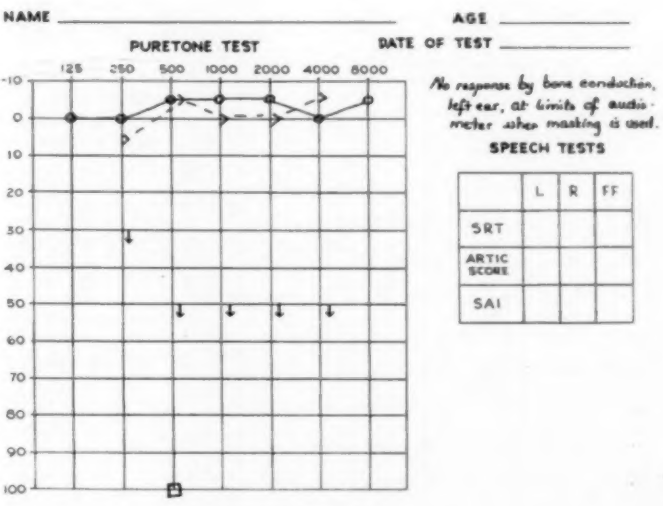
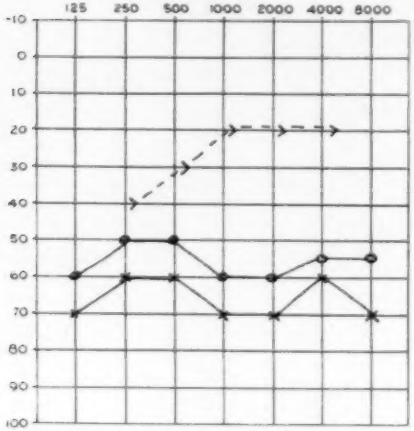

NAME
AGE
PURETONE TEST
DATE OF TEST
125 250 500 1000 2000 4000 8000
-10 0 10 20 30 40 50 60 70 80 90 100
No response by bone conduction, left ear, at limits of audiometer when masking is used.
SPEECH TESTS
L R FF
SRT
ARTIC SCORE
SAI

Fig. 4.

NAME D.A. AGE 12-5-50

PURETONE TEST DATE OF TEST 9-12-55



PGSR

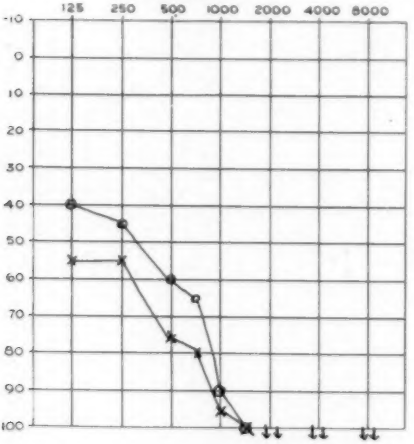
SPEECH TESTS

	L	R	FF
SRT			
ARTIC SCORE			
SAI			

Fig. 5.

NAME D.A. AGE 12-5-50

PURETONE TEST DATE OF TEST 5-9-57



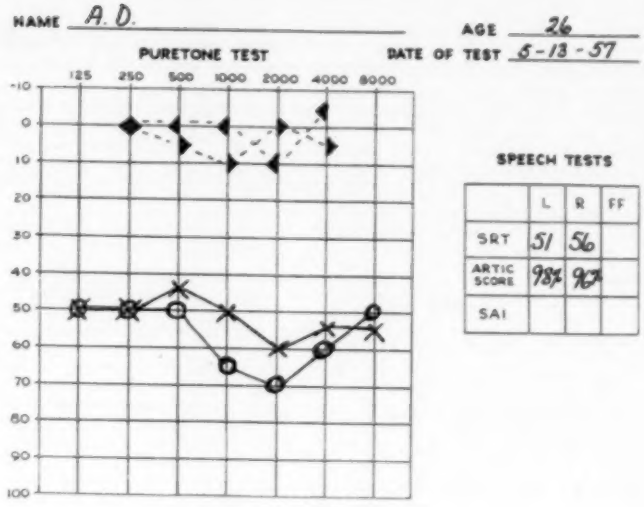
[No response by bone conduction within limits of audiometer]

SPEECH TESTS

	L	R	FF
SRT	75	65	70
ARTIC SCORE			
SAI			

SRT with aid : 25 db.

Fig. 6.


NAME A. D.
AGE 26
PURETONE TEST
DATE OF TEST 5-13-57
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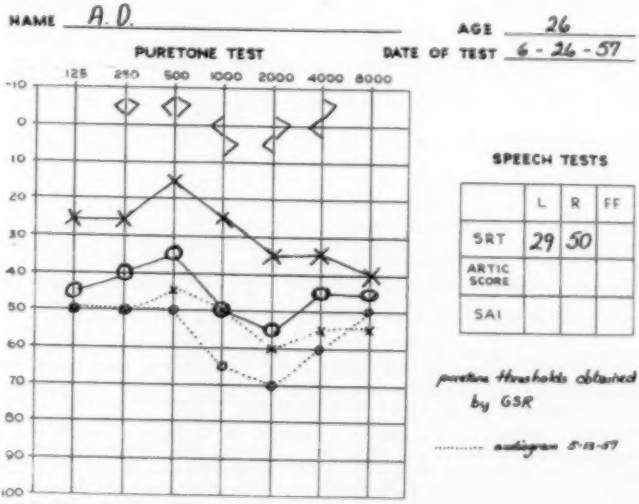
SPEECH TESTS
L R FF
SRT 51 56
ARTIC SCORE 98% 96%
SAI

Fig. 7.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AGE 26
PURETONE TEST
DATE OF TEST 6-26-57
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-10 0 10 20 30 40 50 60 70 80 90 100
SPEECH TESTS
L R FF
SRT 29 50
ARTIC SCORE
SAI
puretone thresholds obtained by GSR
audiogram 5-13-57

Fig. 8.

IV. Collapse of Ear Canal Due to Headset Pressure:

Fig. 7 shows consistent puretone and speech tests in a 26-year-old girl whose response to spoken voice was somewhat better than her audiogram. PGSR testing (see Fig. 8) suggested usable hearing in her left ear.

Stapes mobilization revealed otosclerosis in the right ear with a gain of 32 db. in this ear *after* she was tested by holding the headset a fraction of an inch *away* from her ear (see Fig. 9). A similar test on her unoperated ear revealed a gain from 51 to 29 db. This phenomena, which has been described by Scott Reger is now called the Viehweg syndrome in our laboratory, after the audiologist who discovered two such cases in recent months.

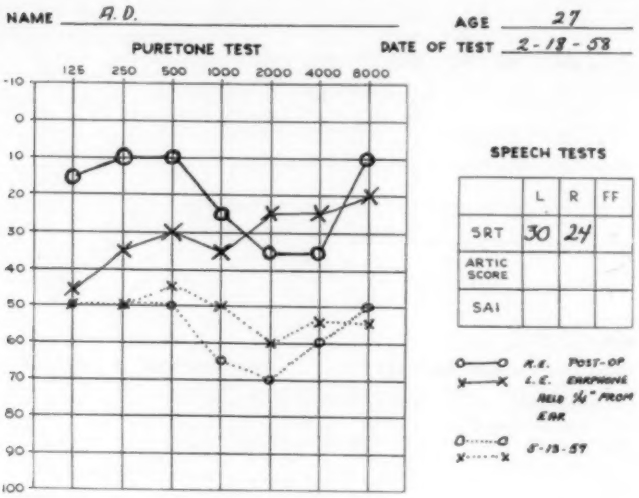


Fig. 9.

MODERATOR HOUSE: Dr. Scheer was not able to attend the meeting. I will now call upon Dr. Shirley Baron.

SHIRLEY H. BARON, M.D., (San Francisco, Calif.): Two years ago in the discussion following the symposium, I presented two cases; one demonstrating the advantages, and one representing one of the possible disadvantages of direct footplate mobilization.

(Slide). This was the case I presented as the one showing the disadvantages of footplate mobilization. This patient had violent vertigo immediately following surgery. The vertigo persisted for four days. There was some loss of balance or dizziness for about six weeks after surgery. I presented this same slide, showing, No. 1, the black line, which was the pre-operative level of hearing, No. 2, the green line, the

immediate postoperative level of hearing and No. 3, the red line, the postoperative level of hearing after two months, showing an improvement in the conversational range, but a sharp drop in high tones; this suggested possible damage to the inner ear by the footplate manipulation. When I returned home from this meeting, about a month later, I received a call from this man in which he stated that his hearing had completely disappeared in the operated ear. Audiograms were done and confirmed that there was a complete loss of hearing (slide).

Now in this case a hole was inadvertently made in the center of the footplate. What, then, might happen if one uses the technique as demonstrated by Dr. Shea in his motion picture, in which it appears that a hole is intentionally made in the center of the footplate? An occasional "dead" ear is bound to result: furthermore, alterations in the membranous labyrinth, as illustrated in the following cases, may occur.

In two patients, additional complications of direct stapes footplate manipulation were revealed in subsequent labyrinthine fenestration operations. In one case (slide), when the cupola was removed, the membranous canal was shriveled into a brownish, flat thread hugging the upper margin of the bony canal; in the other (slide), a thin, brown membrane suspended from the margins of the fenestra like a hammock dipping into the cavern of the bony canal, was present. The status of the membranous canal below this was not determined, as this thin coagulum was not disturbed. Fortunately, both of these patients had a good hearing result, fortuitous no doubt, when one considers what was found in each instance. As Dr. Meltzer has stated, the patient usually is told that if his stapes operation fails, he still has the opportunity of gaining hearing by a subsequent fenestration procedure. In the light of the experiences of these two cases, one must be conservative in footplate manipulation to permit the maximum chance of success for a possible fenestration operation. This should be a cautious, circumferential manipulation of the footplate instead of a hole-punching type of procedure.

Two years ago I stated that it was unwise to proceed with a labyrinthine fenestration at the same operation if it appeared that the stapes mobilization was a failure. Nothing has occurred in the past two years to cause me to change this opinion.

MODERATOR HOUSE: I would now like to call on Dr. Schuknecht.

HAROLD F. SCHUKNECHT, M.D., (Detroit, Mich.): I will limit my discussion to a brief description of my present technique and to an explanation of why I believe it is a rational approach. It should be evident to everyone by now that it is not an easy surgical procedure and that it is not free of complications. In about 400 operations, I have three patients with near total deafness in the operated ear. One of these resulted from simple trans-incudal mobilization, one from chisel mobilization and one following the use of the metallic stapes prosthesis.

I agree that patients must be told that there is a danger that the hearing can get worse. The results are good enough nonetheless to justify recommending the operation after properly informing the patient of possible complications and the chances for success.

I use the direct footplate approach using the Heermann chisels. In my opinion, these chisels are best used by holding them in the fingers. Light to heavy hammer blows are given as necessary to go through the otosclerotic focus when possible. If that fails, an incomplete footplate mobilization is accomplished.

In about 3 per cent of cases, the oval window is filled with a plaque of hard otosclerotic bone, and a successful mobilization cannot be accomplished. With the direct footplate techniques, the footplate is sometimes comminuted, the bluish, thin portion is in pieces, and the crura make contact either with these fragments or sit in a pool of perilymph. The columella effect remains and a good result is possible.

Eighteen months ago, I performed a repeat operation on an unsuccessfully mobilized ear and replaced the previously fractured stapes with a tantalum wire prosthesis. This patient was not a candidate for fenestration surgery because of poor bone conduction thresholds. The tantalum wire was looped and tightened onto the lenticular process with the other end protruding through the remaining pieces of footplate into the vestibule. The patient acquired a good hearing improvement which has been maintained to the present time. Since then, I have used this technique on about fifty patients.

Whenever the direct footplate approach fails to result in a satisfactory columella effect because of crural injury, the tantalum or stainless steel "pin" can be used to reestablish the "columella." Often I remove the head, neck and crura of the stapes. The pins are made of .005 inch suture wire, shaped into a small "question mark" of suitable size, with or without a small "ring" at the bottom. The length of the "pin" is such that when the loop is tightened on the lenticular process, the distal "ring" is in contact with the fragments of footplate.

I now use the pin in over half of all mobilizations and am convinced of its efficacy.

MODERATOR HOUSE: Now I should like to call upon Dr. Fred Guilford.

FREDERICK R. GUILFORD, M.D. (Houston, Texas): We do not believe it is possible with our present knowledge to classify a stapes as to its possibility for mobilization, by the appearance of the otosclerotic focus present. It has been our experience that the examination of the otosclerotic focus alone during the stapes mobilization operation, with our present methods using magnification of from 6 to 25 power, does not give us adequate information concerning the possibility of its mobilization.

The relationship of the plane of the otosclerotic focus to the plane of the footplate, as well as the extent of the otosclerotic involvement, are important factors which may govern the possibilities of surgical mobilization of the stapes.

The relationships of the otosclerotic focus to the level of the footplate may be given a surgical working classifcation as follows:

1. Superficial focus: The main body of the focus is above the level of the footplate. Impingement of the focus on the anterior crus or onto the superior surface of the footplate may be the cause for the fixation. A focus of this type may be quite large and yet the stapes can be readily mobilized. At times the whole superficial focus mobilizes with the stapes.

2. Interstitial focus: In this type of involvement, the fixation is due to a focus which apparently grows into the substance of the footplate without marked overgrowth of bone above the level of the footplate. In this type the white otosclerotic bone can be seen extending from the periphery into the blue-gray footplate, often giving the appearance of the gray-white arcus senilis seen in the cornea. This type of fixation is often strong and rigid. Mobilization effected in the presence of this type of involvement may be associated with a linear fracture of the footplate parallel to the margin otosclerotic focus in the footplate.

3. Deep focus: Fixation of the stapes may be due to a deep focus which has grown onto the under surface of the footplate. No visible evidence of the otosclerotic focus may be present with any magnification when viewed from our surgical approach, yet the stapes may be fixed to varying degrees of slight to marked as judged by the application of pressure to the head or footplate of the stapes.

4. Combined foci: Any or all of the above types of foci may be combined, depending upon the extent of the otosclerotic involvement. The combined type is usually massive in character and usually results in firm fixation which does not yield to mobilization with our present technique.

It is our belief that appropriate attempts should be made to mobilize all fixed stapes in acceptable candidates, regardless of the appearance of the otosclerotic focus. If mobilization techniques fail, or if refixation occurs, acceptable candidates are then encouraged to have fenestration surgery.

MODERATOR HOUSE: Now, Dr. Sullivan.

JOSEPH SULLIVAN, M.D. (Toronto, Can.): We have been stampeded and somewhat mesmerized; however, I congratulate the panel on its frankness and honesty, particularly the summation of the remarks of the panel by Dr. Meltzer. I think that the remark of Merle Lawrence will be validated in the future. Coupling that remark with the excellent contribution of Dr. Hough, and to which Dr. Sooy referred today, this procedure may resolve itself into an approach of congenital malformations of the middle ear rather than the moderately severe otosclerotic.

From the Epistle of St. James, the 1st Chapter, Verses 19, 20, 21: "Let every man be swift to hear; slow to speak and slow to write."

I believe I must emphasize that latter phrase briefly. I make a plea for a uniformity in terminology, correct in basic English, namely, stapes mobilization with a mental reservation that this term may not be entirely accurate.

Three surgeons, Dr. McAskie, Dr. Smith and myself, have evolved a technique and this was written, before the panel remarks came, for stapes mobilization, which in a series of about 500 cases is yielding a 30 db. or better level of hearing in about 40 per cent of the cases for one to four months following operation, for cases suitable for fenestration. There are exceptions; they are few in number. Roughly half of all our mobilization cases fail to achieve a hearing improvement comparable to fenestration, while about 3.5 per cent of all mobilizations end up with a further hearing loss of significant degree.

We believe four factors should be emphasized in this procedure:

1. The use of the operating microscope is essential for the best constant result and there the most important question has been answered, which I had intended to interject to the panel: can a fenestration always follow a non-successful stapes mobilization?

2. Direct visualization of the footplate is necessary, and I hereby insert a word of caution. In spite of what has been said, after nearly 30 years' experience in temporal bone surgery, that footplate perforation is not a fenestration, it is a primary procedure. It is frequently followed by failure and early closure, and is not without danger. Would someone on the panel venture an opinion pertaining to the question about the persisting vertigo, and is labyrinthitis in such a patient a condition where revision of the patient may be attempted?

The use of pressure directly inwards towards the vestibule should be the principal attack in attempting mobilization and it is used only on

the footplate or crura at the very beginning and the end. We test the mobility of the stapes by the lightest pressure on the incus, and that sense of touch can be very well developed in animal experimentation on nerves. Working on either side of the anterior crus, if possible is preferable, not through the otosclerotic focus, because this region has a tendency to fracture.

Finally, a few words of caution concerning the facial nerve. There may be a dehiscence in the facial canal as it runs along the upper margin of the oval window so that the unprotected nerve sheath bulges in the oval window. To quote Cawthorne, there is plenty more to do, and whether to push it or rock it, or pull it, or perforate it, or remove it and bypass it, the stapes deserves all the critical attention we are able to devote to it.

In conclusion, there are many arts among men, the knowledge of which is acquired by experience. For it is experience that causes our lives to move forward by the skill we acquire while the want of experience subjects us to the effects of chance.

MODERATOR HOUSE: I am sure that these thoughts are likewise those of the panel. Now we have had several who have indicated their desire to discuss this. We have in mind also to call on a few more who are not sure at the moment that they are going to discuss it, who have not turned in their cards. First of all I will call on Dr. Theodore Walsh.

THEODORE E. WALSH, M.D. (St. Louis, Mo.): There are just one or two remarks I would like to make. I agree with Dr. Meltzer emphatically when he remarks about being honest with the patient and in telling the patient that he can not always have a fenestration if a mobilization fails. I agree that usually one can do a fenestration when a mobilization fails but not always.

I had a case which illustrated this to my great sorrow. A girl who was an ideal candidate for fenestration, a nurse, who needed her hearing, consulted me some three years ago. She refused fenestration at that time and recently came back demanding the stapes mobilization. She asked me, "Suppose this fails, can I have a fenestration?" and I said, "As a rule, yes, but accidents can always happen, and we might not be able to do it. I still advise a fenestration."

She insisted on the mobilization, and we did it with the very greatest of ease. This was an extremely easy case, and the hearing on the table came up to the 15 decibel level; however, when I removed the drapes, feeling very pleased with myself for having mobilized her stapes, she remarked that she was very dizzy.

She had no nystagmus but did remain extremely dizzy, vomiting and unsteady on her feet, and three weeks later had a completely dead ear. She has not forgiven me.

I believe one should be very careful in all the work one does around the footplate. It is very nice if one can mobilize a stapes easily, but if there is any chance of damaging the hearing by excessive efforts at mobilization, I believe one should quit and do a fenestration later. Incidentally, I agree with Dr. Sullivan when he says that the hearing results from mobilization are no better than they are from fenestration. In reviewing all our cases of both fenestration and mobilization there are very few mobilizations that have a residual hearing level better than

those that are obtained from fenestration, and the percentages of success from fenestration are infinitely higher.

MODERATOR HOUSE: I would now like to call on Dr. Moses Lurie of Boston.

MOSES H. LURIE, M.D., (Boston Mass.): I think this is the most stimulating discussion that I have heard on mobilization of the stapes; but as I listened, I suddenly became aware of the fact that it was not mobilization of the stapes they were talking about. They were talking about various operations on the vestibular part of the membranous labyrinth. Each man seems to have had his own idea how to approach and how to play with it. Until we get a standard method upon which we can all agree, we are never going to get any statistics. We must know exactly what each man does, and we must give each man's variation of the operation a definite place in this work. There is no question in my mind that mobilization will find its place. It is just like the arguments that came up with fenestration. I know all the vicissitudes and variations which we went through, and now are going through again.

I heard some statements which upset me a little bit. One was, if you are going to do bilateral mobilizations of the stapes, wait four months. I would wait a much longer time. I would wait over a year, because you still have the possibility that you may give this patient bilateral deafness, and one must always respect the sensitivity of the membranous labyrinth.

I do not think the criteria of success are definite enough. One talks about four months, two months, three months. No case is a success in my mind, even in the fenestration cases, that does not have good hearing at the end of one year. After a year—then you can start talking about success for mobilization. The one thing I would like a good many of you to carry away with you today is, as Dr. Meltzer said, that you must practice this type of operation. I started to work with it and gave it up because I found that I could not accomodate my eyes to this type of work, but there is no reason why the younger men cannot do it and carry on.

The other thing I would stress is the demonstration that Dr. Lawrence has given you about what the drum and the ossicles do in regard to transmission of sound. Remember, cracks in the footplate may give you good hearing for a short time; but you are playing with a pathological process which you have not stopped and which may destroy all your effort in one or two years. Then the patient will be back to where he was originally.

MODERATOR HOUSE: Thank you, Dr. Lurie. I should like to now call on Dr. Tonndorf.

JUERGEN TONNDORF, M.D., (Iowa City, Iowa): When Dr. Rosen asked me whether I would like to discuss the principles involved in footplate fenestration, I did not know what my good friend, Merle Lawrence, had to say on this subject. Now I feel a little embarrassed. All that remains for me, after his lucid presentation, is to amplify some of the points.

In trying to bring the acoustic task of the middle ear upon a simple denominator one might say that this task is twofold: first, impedance matching; second, preservation of the phase difference between the two cochlear windows. The latter, for reasons of cochlear mechanics, has a value of 180°; however, I would like to point out in this connection that the way nature has solved these two tasks is by no means the only

way this can be accomplished. I would illustrate this point with the aid of three slides.

(Slide). This first slide shows a schematic diagram of the normal middle ear. Impedance matching is accomplished by application of the transformer principle: the large ear drum is coupled to the small footplate via the ossicular chain. In the lower drawing a different approach is used, one which is widely applied in engineering, the so-called acoustic horn. This horn accomplishes essentially the same that the ossicular chain does in the upper drawing. I admit that, within the limited space between the pinna and the window, a horn would be somewhat unsatisfactory at low frequencies, that is below 500 cycles.

(Slide). Now we come to the second task: the preservation of phase differences. In this slide I made up two theoretical cases which represent two extreme solutions of the problem. The first of these solutions consists in doubling the ossicular chain. Normally (the upper chain of this drawing), the eardrum and the oval window move in phase. In the lower (hypothetical) chain, the incus is assumed to pivot about its center so that the eardrum and the round window, to which this second chain is connected, move in opposite phases. This way one would obtain a drive mechanism which engineers, by a very descriptive term, call a pushpull drive mechanism.

The other extreme solution would consist of shielding the round window completely so as to produce an infinite impedance mismatch between the eardrum and the round window. From the third drawing on this slide it is obvious that Nature does not use either one of these extreme solutions but makes somewhat of a compromise. First, there is a certain amount of phase shift through the ear drum. This shift is maximal, namely 90°, at 1500 c.p.s. and smaller for all other frequencies, being positive at lower and negative at higher frequencies; thus the phase shift principle is operative to a certain extent. The second principle is the impedance mismatch. The ossicular chain represents a solid coupling system which has a high degree of energy transfer. The other pathway, eardrum to round window, has a lower degree of energy transfer; furthermore, the two windows are of different size, the round window, being the smaller of the two. Third (not shown in this schematic drawing) the eardrum and the round window are not in an identical plane so that partial reflection might occur. Of course, this latter fact does not operate at low frequencies, but it may become important, however, at frequencies above 2,000 cycles.

(Slide). This last slide gives the reason why Dr. Rosen's method of footplate fenestration is acoustically feasible. Now both windows are coupled to the eardrum in a similar fashion; however, because of the fact that the window in the footplate is so much smaller, we have a better impedance match from the ear drum to the round window and a lesser impedance match to the window in the footplate. The assumption appears logical, as Dr. Lawrence has pointed out before, that the relative sizes of these windows are very critical. Without having done any measurements or experiments along this line, I would venture to predict that a plot of amplitude of window motion vs. size of the footplate window would first increase and after going through a maximum, it would go down again. At this time, I am in no position to say anything about the degree of gain one might achieve by this method, and of course it is completely out of my province to say anything about the probability of postoperative changes. The only thing I would point out here is that this method is acoustically feasible.

MODERATOR HOUSE: Dr. Day, may we have your comments?

DR. KENNETH M. DAY: I would comment on the comparison of the hearing following fenestration and mobilization. There must be a dozen patients upon whom I performed a fenestration some years ago and have recently mobilized the stapes of the other ear. In half of these cases the hearing level in the fenestrated ear and in the mobilized ear is almost the same.

One of the patients happened to be a young musician. He has music in his fingers. Discrimination scorers are the same, but he said, "I like the first ear for music." There is a little distortion in his mobilized ear. Most of the patients upon whom I have done a mobilization on the second ear say "I may hear as well in the mobilized ear, but I seem to hear more clearly in the fenestrated ear."

Is that due to traumatic labyrinthine reaction from working around the footplate? We are always worried about trauma when we make a fenestra in the horizontal canal. I wonder if this is not a case of more traumatic reaction in the cochlea by working on the footplate than back in the horizontal canal.

MODERATOR HOUSE: Now we will hear from Dr. Hoople.

GORDON D. HOOPLE, M.D. (Syracuse, N. Y.): This has been most interesting. There are many things that one could say, but I want to confine my remarks to one aspect of the whole subject and that is none of us should go away from here thinking that we have the final answer on the chances of percentage of success. Dr. Kos has told us the reasons why. There is a figure projected which shows 42 per cent as the overall results of all the panelists. If I interpret the presentation correctly this is the result of all their cases to the present time. It includes some of the cases that were not successful in the very beginning of mobilization surgery. I am afraid that it also includes some cases which the panelists have done by footplate manipulation which in three months, six months, a year or more from now may not have as good hearing as they have reported at the present time.

I have some evidence, which is not ready for presentation now, that footplate work is spoiling some of the results of men who did not formerly attack the footplate. One member of the Society who has spoken this morning, stated at Philadelphia at the Midwinter Meeting that he had 72 per cent successes in doing mobilization surgery but in four months 17 per cent of these had regressed to the former hearing level. (These are his own figures. Maybe I have missed out by one or two per cent but that is not the point.) I am not belittling him at all but this is success of an operation but not success in producing better hearing as far as the patient is concerned. So we will have to wait a long time before we know what the results are, and we must take into consideration the present rash of footplate manipulations. I use that word advisedly.

Finally I would like to say one thing which I think most of us have forgotten. The last word that was said in the panel two years ago in Montreal was said by the man who today would be the President of this Society, had he lived. He, himself, was not a fenestrator, but he said this: "I believe that this work should be done by men who know fenestration surgery." I leave you to put whatever interpretation you wish on his words but I want to remind you that he said them.

MODERATOR HOUSE: Now with the passage of time I take this opportunity to return to the panel for any closing comments they might like to make.

DR. LAWRENCE: I don't believe that there is any need for saying three times what I said, but I thank Dr. Tonndorf for the amplification and would like to re-emphasize what the result will be, if one can re-establish the normal condition. I leave the rest to the panel.

MODERATOR HOUSE: Dr. Goodhill, I would like to ask you two questions: one is in answer to Dr. Sullivan. Can cases of persistent, annoying vertigo be revised?

DR. GOODHILL: I have not revised any. In answer to Dr. Sullivan's question as to why they happen, I am quite sure that they are due to permanent displacement of utricle, saccule, macula and fixation, by some kind of fixation in the vestibule.

MODERATOR HOUSE: Are there any other closing comments you would like to make?

DR. GOODHILL: I would like to make one comment in regard to Dr. Hoople's statement. I think he would be very happy to learn of the activities of the Committee of the Otosclerosis Study Group, which met here a day or two ago. I don't believe I am going to disclose any confidences when I tell him that this committee, working up a report for the next meeting in October, has decided to eliminate three words in their recommendations for evaluation techniques for all types of conductive deafness surgery. The first word is success; the second word is permanent, and the third word is total. It will be our recommendation that neither of these three words be used in any reporting techniques.

It will be our further recommendation that all evaluations be reported in two separate categories, each category being reported for Class A, Class B and Class C cases. The first category will be the 30 db. level. This is important from the psychosocial point of view. This is one method. The other method will be evaluation in terms of the degree of closure of the air-bone gap, and the word which we probably will use will be per cent improvement, or per cent closure, of the air-bone gap. The word "success" is out.

MODERATOR HOUSE: Thank you Dr. Goodhill.

I should now like to ask Dr. Shambaugh for any closing remarks, but before doing so I would like to ask him in view of our techniques what are his thoughts pertaining to mobilization of a patient who has been previously unsuccessfully fenestrated on the same ear.

GEO. E. SHAMBAUGH, JR., M.D., (Chicago, Ill.) (Closing): We have attempted to do this procedure in a number of cases and all of them were failures. Theoretically such a procedure, if you can also perform a myringostapediopexy might be successful but in none of the cases that we have done it has there been any improvement. So we feel that this procedure is not of any value.

In closing, I would like to show a single slide, if I might. This slide emphasizes some of the fundamental physiology of the ear which Dr. Lawrence presented so beautifully. In the upper left see the patient's air-bone. His is the type of case classified as Type C, because of poor bone conduction but with a good air-bone gap. The patient had mobiliza-

tion by one of my associates in which the crura were fractured at the same time the footplate was mobilized, and you will notice the further loss of hearing by air, and the increase in the air-bone gap, due to discontinuity of the ossicular chain, despite a successful mobilization of the footplate. Two years later the patient returned for revision. At this revision a tantalum wire, as Dr. Schuknecht has described so nicely, was placed between the incus and the center of the mobilized footplate and we see that the air-bone gap now has been reduced to somewhat less than 50 per cent of the pre-operative level. We have re-established the continuity of the ossicular chain and, as Dr. Lawrence has stated three times, I think, I would like to say for the fourth time: for best results we must have a continuity between the tympanic membrane and the movable portion of the footplate.

MODERATOR HOUSE: Thank you, Dr. Shambaugh. Now may I call upon Dr. Kos for any comments he might have in closing.

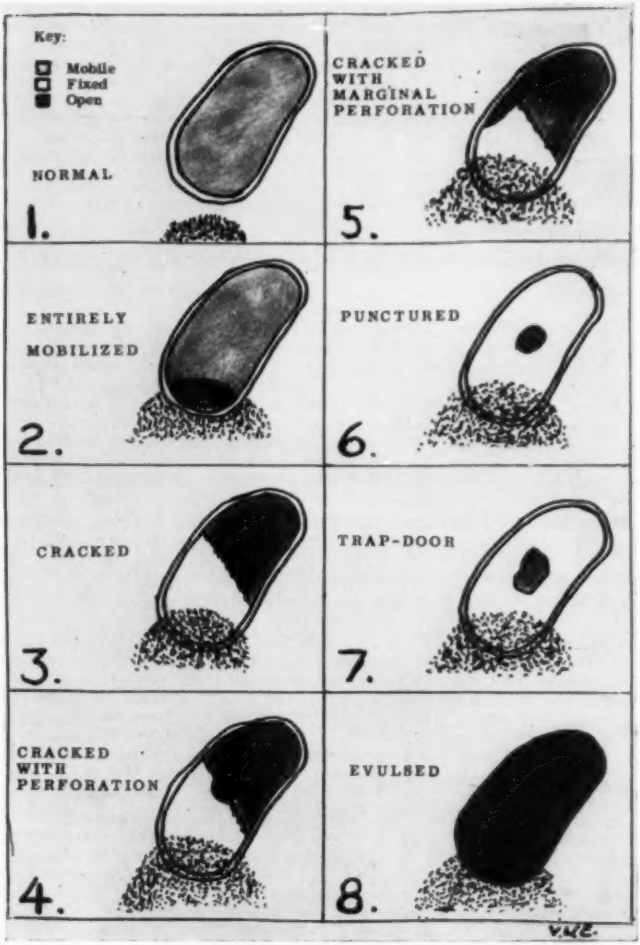
C. M. KOS, M.D. (Iowa City, Iowa) (Closing): Well, the figures speak for themselves, though not very accurately for obvious reasons which have been mentioned here several times. I should like to emphasize, that regardless of any other concept in connection with this operation, at the present time it is essentially an exploratory operation. It is exploratory for two principal reasons—One is that there is no pre-operative or clinical clue on which one can hang enough confidence to predict what the outcome may be; second, there is no surgeon who can possibly accurately anticipate what he will find before he raises the tympanic membrane.

MODERATOR HOUSE: Now Dr. Fowler, before closing I would like you also to answer the question of Dr. Sullivan, namely to comment a little further on persistent vertigo following mobilization surgery as a complication.

EDMUND P. FOWLER, JR., M.D. (New York, N. Y.) (Closing): I think it is wise at this point to emphasize that the footplate can be mobilized in a number of ways. This is my personal opinion, and not held by some on the panel and that is why I left it to this time. I believe that as a rule the stapes (Slide 1) is mobilized by fracture through the footplate as shown in I, IV or V. I also think that this is probably the best way to mobilize the stapes ankylosed by otosclerosis because if it is mobilized through the focus as in II, it seems to be the ideal way to invite more closures.

(Slide 2). To answer Dr. Sullivan to a certain extent, this is a section of the footplate of a patient in which an anterior crurotomy was done. Inadvertently, in testing the footplate a puncture of the footplate occurred (between the arrows). This patient died 15 months after the operation and you can see that there is still a spicule of bone in the labyrinth (above arrow on left). There was no infection in the labyrinth. I think that if you push this spicule of bone a little further that it can be pushed into the utricle or it can rest on the utricle and produce vertigo by acting as an overgrown otolith. Unfortunately we are not always as lucky as we were in this case when there is a hole in the footplate of this type. If there is any infection present in the middle ear it could get into the labyrinth through such a hole.

Finally, I would say that the size of the hole in the footplate may make a great deal of difference as far as subsequent hearing is con-



Slide 1. Diagram of eight ways in which the perilymph might be mobilized in various types of mobilization procedures.

cerned; also the stiffness of the healing process. Months later it can even be what the stiffness is later, which produces the effects that were mentioned by Dr. Lawrence and Dr. Tonndorf. Finally, the position and overhang on the round window may also make a difference. Until we understand these things we will not understand mobilization.

Slide 2. Histological preparation of a patient who had stapes mobilization (anterior crurotomy and footplate puncture) 15 months before his death. Fractured anterior crus is shown just behind the classical otosclerosis on the left and the fibrous closure of the hole in the footplate is seen between the arrows. Above the arrow on the right is a spicule of bone which has dropped downward into the vestibule, and below the arrow on the left is a bit of floating endostium. Note that there is no evidence of infection in the labyrinth.

MODERATOR HOUSE: It is now my pleasure to make a few closing remarks on the subject.

First, Dr. Lawrence has brought to this group his comments and description as to why these operations work. He has also made the prediction that in the eighties we will return possibly to the fenestration operation in otosclerosis. I would like to comment that the fenestration operation still offers the very best chance of restoration of hearing in suitable cases of surgical otosclerosis.

Dr. Lindsay pointed out by means of histopathological examination why footplate techniques are essential in some

extensive cases of otosclerosis. I would like to make a plea for all of us to supply these gentlemen doing histological work with more and more specimens.

Dr. Goodhill confirmed our indications for the initial operation as presented two years ago. There has been little change in these indications. He stressed the necessity of unilateral masking to prevent the operation of stapes mobilization being done on unilateral perceptive deafness. He pointed out the indications for revision. He stated we are not in agreement regarding indications for revision. The greatest help in this regard is to observe and make accurate detailed notes as to what was done at the initial operation. Note at that time as to whether or not you feel a subsequent revision is indicated.

Dr. Shambaugh told us that the techniques have changed. These changes include the use of the microscope, audiometry in the operating room, and more direct approaches to the footplate itself. Both Lawrence and Shambaugh have stated that small perforations in the vestibule may improve hearing, but the lasting results of these procedures are questioned at this time. He pointed out that the fenestration operation is predictive. The frustration operation (stapes mobilization) is not predictive.

Dr. Kos told us that at Montreal this panel had operated approximately 1,100 cases. Today this panel has operated 7,400 cases. The results he reported average two months following surgery and he reported that 15 per cent of this group had subsequently regressed. He mentioned that 49 per cent of our cases received improved hearing. Of these 49 per cent who obtained improvement, 42 per cent reached the 30 db. level or better. It is the opinion of the panel that the incidence of regression is greater as we work more and more on the footplate.

Dr. Fowler stated that three per cent of the cases operated by this panel were worse, of which one-half of one per cent were severely worse. Included in this series were three dead labyrinths that occurred following stapes mobilization. He also mentioned that others have reported two deaths and one extradural abscess following this surgery.

The comments of Dr. Meltzer are always highly valued for their conservatism and the prospectives of the future are well taken. We grow with otology, with experience.

To Dr. Rosen we are grateful for his observations that have brought about this fine discussion today. We are also grateful to him for having pointed out that this operation is not a simple procedure, but indeed a complex one and not without the possibility of significant and serious complications. Dr. Rosen stated his experience with fenestration of the footplate has been gratifying in obtaining immediate as well as lasting improvement in hearing. This was not the opinion as expressed by Dr. Shambaugh. Only time will settle this difference of opinion.

In conclusion I would say that we must continue with thoughts of conservatism and an open mind. Great progress is being made in all phases of otology through laboratory and clinical research. The progress being made in stapes mobilization surgery has been presented today.

I thank the panel, the official discussers, the other discussers for their contributions today, and the audience for your kind attention.

AMERICAN BOARD OF OTOLARYNGOLOGY.

The American Board of Otolaryngology will conduct only one examination in 1958, and this will be October 6-9, 1958, in Chicago, Illinois, at the Palmer House.

For further information address Dr. Dean M. Lierle, Secy-Treas., University Hospital, Iowa City, Iowa.

JULIUS CAESAR AND THE FALLING SICKNESS.*

TERENCE CAWTHORNE, F.R.C.S.,

London, England.

The idea for this paper came when, some years ago, I was re-reading Shakespeare's "Julius Caesar" at a time when I was preparing an article on Mèniére's Disease. In the very same scene, and separated by but a few lines, the following statements are made:

Julius Caesar (who has been talking to Anthony about Cassius, ends by saying): "Come on my right hand, for this ear is deaf."

Then shortly after, in a discussion about Caesar between Cassius, Casca and Brutus, the latter says:

"'Tis very like he hath the falling sickness."

Now it has always been assumed that Julius Caesar suffered from epilepsy, but unilateral deafness and the "falling sickness," I thought, could mean Mèniére's Disease; so ever since then I have been inquiring into the life and times of Julius Caesar, with particular reference to his ailment.

As the 2,000th anniversary of his death by assassination fell on March 15, of last year, it seems an appropriate time to discuss his disorder and to recall his remarkable career, which was brought to a brutal end by a determined band of his associates, many of whom he regarded as trusted friends. Though he was the most powerful and the most popular man in the civilized world, and though it was freely admitted that he had done more for the glory of Rome than any other citizen, he had by his remarkable and versatile talents shown up the lack of ability in many others; earning the envy and malice of those around him whose ambitions were so much greater than their often meager abilities.

A Patrician by birth, his was one of the oldest and noblest Roman families, the Julians, and when his aunt Julia, who

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had married the Democratic or Popular dictator, Marius, died, he said in the funeral oration:

"On her mother's side Julia descended from the ancient Kings; on her father's from the Immortal Gods Themselves. Her mother and my grandmother, Marcia, descended from Marcius, the fourth King of Rome; while we of the Julian House trace back our origin to Venus herself. In our family, therefore, we combine the Divine Right of Kings, who are the greatest among men, and the worship of the Gods, to Whose Power even Kings must bow."

Though obviously proud of his family and possibly not unmindful of his destiny, he was no idle boaster when it came to his own deeds as set out in his "War Commentaries." It could never be said, however, that he hid his light under a bushel; indeed, it would have been difficult so to do, as his great deeds would have shone out.

In view of his noble origin, which included nearly all the Patricians, it might be thought that Julius Caesar would follow the Optimates, or Republican Party, which included nearly all the Patricians. Instead he joined their opponents, the Populares, or Democratic party, to which the middle classes gave allegiance. In this, no doubt, he was influenced by his uncle, Marius, who had been the leader of the Populares. After the eclipse of Marius when Julius Caesar was a youth, Sulla, the champion of the Optimates, came to power and dealt very severely with his political opponents. Fortunately for the Roman Empire, the young Julius was left alone, though he had to travel abroad for some time.

Despite the great care taken to avoid any individual form of dictatorship in the Roman State, it gradually came about that political power was concentrated into the hands of a comparatively small group of Patrician families who saw to it that they dominated the Senate. Consequently, many of the laws which were passed favored the Patrician and landowning classes. Successful wars brought more slaves into Roman hands, and these slaves went to swell the estates of the landowners who were able to cultivate their land with cheap slave labor. Thus the smaller farmers could not com-

pete with this cheap labor and sold their farms to seek their fortune in the Capital, adding to the number of the "hangers on" and unemployed.

Successive political leaders vied with each other to gain popularity, and the increasing inefficiency and corruption of both parties were undermining the State, so that the time was ripe for a man with that rare combination of ambition and ability to rescue the Roman Republic from the slough of corruption and selfishness which was threatening to engulf it. Though we know that Julius Caesar was the man to do this, it took him 50 years of pushing, bribing, intriguing and fighting to convince his contemporaries. Even when he had succeeded, they could not forgive him, and despite all he had done for them and for the State, they murdered him; though they preferred to call it "tyrannicide."

This was a political blunder, for Caesar was the one man who could have built upon the old foundation, a solid and workable government. His assassination involved the State in fresh struggles and civil wars, until in the end it fell under the supremacy of Caesar's heir and nephew, Augustus, who established a far more despotic rule than that which the so-called liberators had attempted to avert.

Born in the year 102 B.C., Caesar was, thanks to his uncle Marius' influence, given, of all things, a senior religious appointment as Flamen Dialis, at the age of 15. This gave him a seat in the Senate, but precluded him taking an active part in war or politics. This did not last very long, and at 19 he went abroad, gaining experience in war as an aide-de-camp and as an orator and lawyer, from the leading teachers in the Grecian Islands. This included an episode at the Court of King Nicodemus, which later on was magnified by his opponents into a homosexual scandal; however, his conduct in more mature years shows that if this incident ever occurred, it was but an adolescent prank, for Sir Charles Oman refers to him later as "the inevitable co-respondent in every fashionable divorce." Returning to public life in Rome at the age of 30, he achieved the coveted Consulship by the age of 42.

He was not too particular about the means he employed to

reach this post, which has been described as a "carnival of illegality and mob law, which made a fitting close to the whole of his demagogic career."

Once his Consulship was over he took over the pro-Consulship of the Gallic Provinces, at the same time forming an uneasy alliance with the financier, Crassus, and the ex-soldier, Pompey. This was known as the Triumvirate, and each was of equal Consular rank and power. Caesar left for his Gallic Province, and it was 10 years before he returned to Rome, having in the meantime conquered the whole of Gaul, contained the Germanic hordes and invaded Britian twice.

Despite his previously slight military experience, Julius Caesar soon proved to be an exceptional leader in war. He was not only a great strategist and military tactician, but he was also a leader of men. Many was the time that he, personally, led his men into battle, and he was often in the thickest of the fight. As an orator and as a politician he was unequalled, and by sheer force of intellect he could persuade men to his way of thinking, equally well in committee, in the political arena and before a group of tired and even rebellious soldiers. Even the eloquent though vacillating Cicero, the silver-tongued orator, was compelled to admit that he had met his master in Julius Caesar; but he never forgave Caesar for this, and I believe that it was he, Cicero, who was behind the plot to kill Caesar.

At the end of the Gallic campaign Caesar fought his way back to Rome, from which his opponents, the Senate (headed by Pompey) had fled, and four more years of fighting in Spain, Greece, Africa and again Spain were needed before he finally returned triumphantly to Rome.

During the year that was left to him he laid the foundation of a new autocracy headed by a Sovereign to rule the Roman Empire in the place of the Senate and the people. His names have been immortalized in the month of July, and in the title of Caesar, Czar and Kaiser. He forgave his political enemies, and this was his undoing, for they could not forgive him.

As a young man he was undersized and delicate, but he grew up to be virile, tough and even licentious. Though he

loved luxury and power, he never over-indulged in eating or drinking, and between the ages of 40 and 55 lived the hard life of a soldier on campaign. Always in the public eye, any disorder, infirmity or even slight accident is sure to have been noted and even magnified. We have heard of his baldness and of his amours, and even when he stumbled and fell when disembarking onto African soil during the Civil war this was noted by Plutarch. I am sure that if he had been subject to real epileptic fits we would have heard much more about them than has come down to us from the writers of those times.

Our main source of information about Caesar's "falling sickness" comes from "Plutarch's Lives," in which it is mentioned three times.

The first was part of a general description of Caesar:

He was lean, white and soft-skinned, and often subject to headache, and other while to the falling sickness, the which took him the first time, as is reported, in Cordoba.

Now Caesar was three times in Spain. The first time was before he was Consul; the second in the early stages of the Civil War before he defeated Pompey at Pharsalus, and finally after the African campaign, when he was dealing with Pompey's son and his followers. This must refer to the second campaign in Spain, as Plutarch's next reference to the sickness is in the African campaign at the battle of Thapsacus against King Juba and Scipio. When he did not take his usual place in the forefront of the battle Plutarch says:

For as he did set his men in battle the falling sickness took him, whereunto he was given, and therefore feeling it coming, before he was overcome withal, he was carried into a castle not far from thence, where the battle was fought, and there took his rest till the extremity of his disease had left him.

Plutarch's third reference is to the time when the populace offered Caesar the Emperor's crown and he refused it three times:

Notwithstanding it is reported that afterwards, to excuse this folly, he imputed it to his disease, saying that their wits

are not perfect which have this disease of the falling evil, when standing on their feet they speak to the common people, but are soon troubled with a trembling of their body and a sudden dimness and giddiness.

It may be noted that in none of these three statements is there any reference to loss of consciousness, to foaming at the mouth, to twitching of the face or limbs, or indeed to any of the other manifestations of epilepsy.

Plutarch, the Greek Philosopher and biographer, was born in the reign of Claudius half way through the first century, A.D. He wrote in Greek and his most famous work, his "Lives," compares and contrasts the Greek and Roman heroes. His is the most contemporary and the fullest life of Caesar.

There is another by Suetonius, the first in his series of "The Lives of the Caesars." This was written some 70 years after Plutarch. Suetonius had the advantage of being Secretary to the Emperor Hadrian and in this position had access to Imperial Archives. His "Life of Caesar," written in Latin, makes but one short reference to Julius Caesar's ailment: "He was twice attacked by the *falling sickness* during his campaigns."

As might be expected, Caesar himself makes no reference to this sickness in any of his works, nor is it mentioned by Cicero, Lucan or Dio Cassius.

Shakespeare's source of information about Julius Caesar came from Sir Thomas North's translation of "Plutarch's Lives"; and it is Sir Thomas North who coined the term *"the falling sickness."* He did not translate direct from the Greek, but from the French of James Amyot, Bishop of Auxerre. There is no mention in Plutarch, or in any other Greek or Roman author, of Caesar's being deaf, even in one ear.

The only reference I can find of Julius Caesar's deafness comes from George Wherry, in "Notes and Queries (1909), Series 10, Vol. XL, p. 243.

Julius Caesar's Deafness: Shakespeare makes Julius Caesar deaf in the left ear. To Mark Anthony, Caesar says: "Come on my right hand, for this ear is deaf;" and his attacks of

epilepsy are also noted in the play "He hath the falling sickness." It is quite possible that attacks of giddiness, associated with Meniere's Disease of the ear, may have been mistaken for epilepsy. The Romans were familiar enough with epilepsy, which they called "morbus comitialis," from the attacks witnessed in the Forum or Senate House, which broke up the Assembly; and it is unlikely that aural vertigo was understood at that time.

Where, then, did Shakespeare get his reference to Julius Caesar's being deaf in the left ear? It has been suggested that there might be some political significance in this, but it cannot be in the modern sense, for the term "Left Wing" and "Right Wing" became significant only in the Assembly of the French Revolution. Again, could it be that Caesar was suggesting that he was going to forget what had just been said? But as he had just been doing all the talking and very little listening, there is little point in this suggestion. As there does not appear to be any mention of Caesar's being deaf in any of the references available to Shakespeare, he may have put this in so as to add a touch of realism to the part. We know that Shakespeare always made his great historical figures behave like ordinary human beings; and indeed, it is just that homely touch that makes his characters live. It could well be that Shakespeare knew someone, or possibly more than one person, who was troubled with the "falling sickness" and deafness, and that he introduced the deafness in one ear in order to make Caesar like other men. There is no reason to suppose that the deafness was brought in to help the stage business.

Even if we deny Caesar's deafness on the grounds that we have only Shakespeare's word for it, we are still left with his "falling sickness." Plutarch, writing in Greek, refers to this as "Epileptikois," a term used in those days for any seizure that caused the sufferer to fall down and be helpless. It is not surprising that many writers translated this as epilepsy, and so the term "falling sickness" and epilepsy have been loosely regarded as the same thing.

Suetonius, writing in Latin, uses the term "morbus comitialis," the disease which, when it attacked a member of a

committee, ended the meeting. Clearly such a description could include epilepsy, a stroke, a heart attack, and also an attack of Meniere's Disease.

Now, as Caesar's mental and physical powers remained unimpaired between attacks, it seems unlikely that they were caused by heart disease or a cerebral catastrophe. This leaves epilepsy and Meniere's Disease.

When the "falling sickness" first overcame him, Caesar was in the early 50's, a common time for Meniere's Disease to start. As the most famous man of the Ancient World, there was no shortage of writers and poets to publicize his failings as well as his strength. Had he really suffered from epilepsy it is difficult to believe that it would have been passed over in the few words that have come down to us; nor can it be said that the Romans were unacquainted with epilepsy, for here is a description given by Lucretius of an epileptic fit:

Oft times with violent fits a patient falls,
As if with thunder struck; and foams and bawls,
Talks madly, shakes, moves here and there, breathes short,
Extends and tires his limbs with antic sport;

* * * * * *

His weakness wears and he forgets his pain;
His strength, his life, his sense return again.

Had Caesar suffered from major epileptic seizures, surely these sensational attacks would not have been passed over so slightly by the careful biographer Plutarch, or by the gossip Suetonius.

Had these attacks been something more serious than a labyrinthine disturbance, they must have left a greater mark on the man who, for the last 15 years of his life extended and dominated the Roman State. A man, moreover, who, at the end of 12 years' continuous fighting and political intrigue could take time off to share with Cleopatra one of the great love affairs of the world.

That it could have been a minor form of epilepsy cannot be denied, though sufferers from "petit mal" so often are

troubled also with bad tempers, fits of depression, or some form of neurosis; and there is nothing to suggest that Caesar was ever troubled in any of these ways. Coming on at that time of life and not leading to any deterioration of mental or physical powers it is, in the light of our present knowledge, much more likely to have been a benign disorder such as Meniere's Disease.

We are still left with the problem of deafness, and though I have been unable to trace any contemporary account of Caesar's being deaf, I shall always be grateful to the memory of Shakespeare, not only for the plays which we enjoy, but for suggesting an alternative and to me, plausible cause for Caesar's "falling sickness."

ALUMNI ASSOCIATION OF THE NEW YORK EYE AND EAR INFIRMARY.

The Annual Spring Meeting of the Alumni Association of the New York Eye and Ear Infirmary last April was so well received that it has been decided to expand next year's meeting, which will take place from April 20-23, 1959.

Symposia and lectures on Hearing Rehabilitation, Endoscopy, and Ear Surgery will be conducted. It is also planned to offer refresher courses in Mastoid and Fenestration Surgery and Stapes Mobilization Techniques.

More complete information regarding the meeting will appear in a later issue of THE LARYNGOSCOPE.

CASE OF TRACHEO-MALACIA FOLLOWING REMOVAL OF ADENOMA OF THYROID GLAND.*

DEZSO KASSAY, M.D., (By Invitation),

Philadelphia, Pa.

A 50-year-old male, a dentist, had been complaining, for about six months, of a gradually increasing dyspnea and stridor. Bronchial asthma had been diagnosed in a hospital in Hungary.

On admission we found a well nourished man, 5'4" tall, and muscular; the patient's neck was extremely thick and short. On the left side of the neck a spherical resistance with a diameter of about 5 cm. could be palpated. This tumor moved up and down simultaneously with the swallowing action. The dyspnea appeared to be mild and the stridor audible only before the open mouth.

Planigraphy (see Fig. 2-A) showed the trachea displaced slightly to the right, and its lumen narrowed markedly from the left side by a protrusion 45 mm. in length. Tracheoscopy showed the tracheal lumen enormously stenosed by lateral compression. The mucosa appeared red and swollen, but no tumor tissue was noted. The biopsy and histologic study revealed only inflamed mucosal structure.

TRACHEAL STENOSIS DUE TO COMPRESSION BY ENLARGED THYROID GLAND WAS DIAGNOSED.

At external operation (Rubanyi and Kassay) we found a soft, vascular, encapsulated tumor which was about 5 cm. in each dimension; resection of the tumor and trachectomy were performed. The tracheal cartilages on the left side for a length of about 45 mm. were destroyed, and the soft tracheal wall collapsed into the lumen. Adenoma of the thyroid gland was diagnosed histologically. Removal of the tumor did not

*Read at the meeting of the Eastern Section, American Laryngological, Rhinological and Otological Society, Inc., Philadelphia, Pa., Jan. 9, 1958.

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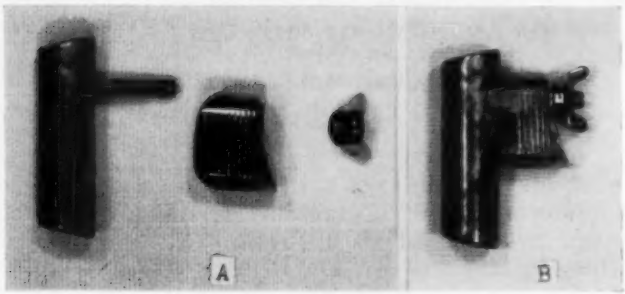

Fig. 1. Parts of the cannula (A) and the assembled cannula (B).

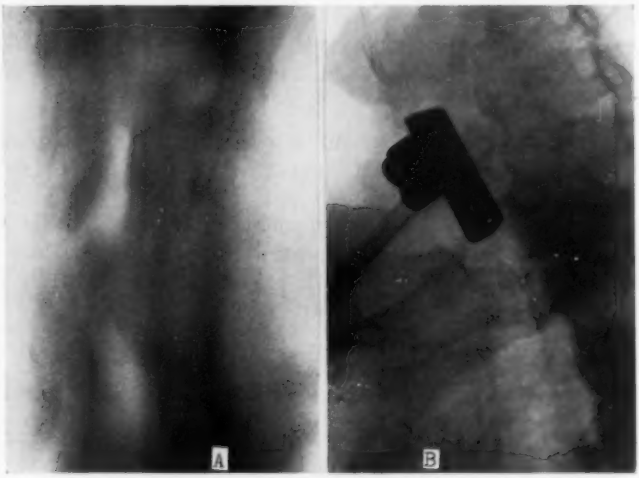

Fig. 2. Planigraphy (A) shows the cervical trachea displaced slightly to the right and its lumen stenosed from the left side by external compression. X-ray film (B) demonstrates the retaining cannula in place. Note the wide lumen of the trachea.

diminish the stenosis; this situation, checked repeatedly by tracheoscopy and planigraphy did not change for three months. The patient refused resection of the tracheal portion, collapsed by tracheo-malacia, or any kind of surgical plastic repair; however, he was willing to wear a permanent tube suggested

by us, which would keep the tracheal lumen open and would not disturb him in his work.

Thus we designed a special tube for him which would hold itself in position, could be easily changed, occlude the stoma well and prevent the outflow or coughing out of tracheo-bronchial secretions. Naturally we created first a longer stoma sewing the tracheal wall to the skin of the neck.

A metal tube (see Fig. 1), with a 12 mm. outside diameter was used. Both ends were cut off obliquely, the anterior wall being longer than the posterior, which facilitated the intro-

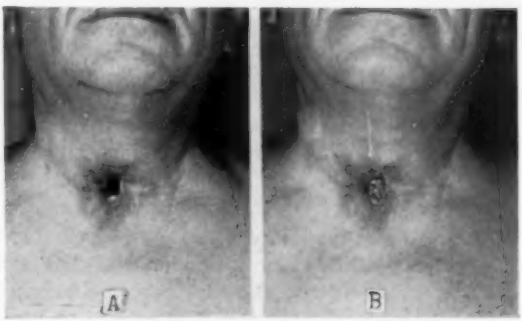

Fig. 3. The open stoma (A) and the cannula in place (B).

duction of the cannula. The total length of the tube was 45 mm. A screw was attached to the anterior wall, 12 mm. from the upper edge of the tube. A metal block, fitted precisely to the stoma and a wing-nut completed the instrument. The block was 20 mm. long and 11 mm. wide, slightly less than the outside diameter of the tube; the depth of the block measured 14 mm. on the top and 16 mm. on the bottom. At the proper point in the block, a hole was drilled for the screw. The distance between the screw and the upper edge of the tube was shorter than the vertical diameter of the tracheal stoma.

At introduction the longer part of the cannula (below the screw) entered the trachea first until the screw touched the lower edge of the stoma, then the whole cannula was slid into

the trachea and lifted, so that the upper portion of the tube entered the lowest part of the larynx. The block was slid over the screw into the stoma and fixed in position by the nut (see Fig. 2-B).

This procedure (the change of the cannula) was performed by the patient himself once or, when necessary, twice a day. The cannula did not injure the tracheal and laryngeal tissues because it followed the movements of the trachea and larynx precisely and kept itself in position without being anchored by any mechanical means except that described above (see Fig. 3). When I left Hungary, more than a year ago, the patient had worn his cannula for six months without any complaint.

After this successful attempt I looked for similar solutions in medical publications, and found only one: Jackson's bronchoscopy, esophagoscopy and gastroscopy, published in 1934. Their retaining intubation tube was used for treatment of chronic laryngeal stenosis. The tube was introduced through the mouth, and then a post was screwed in through the tracheal stoma and a block was slid into the stoma. Similarity of the two methods is the use of screw and block for keeping the cannula in place, in the larynx or trachea. The difference is that the Jackson's introduced the tube by laryngoscopy, which could not be performed by the patient, himself, and thus it would not serve our purpose; however, essentially, we adapted an older method to a newer purpose, which may be expressed by the old Hungarian proverb: "Read wise old books and create new things."

Lankenau Hospital.

SYMPOSIUM: TYMPANOPLASTY.

MODERATOR:

F. A. SOOY, M.D., San Francisco, Calif.

PANEL:

VICTOR GOODHILL, M.D., Los Angeles, Calif.
LELAND R. HOUSE, M.D., Los Angeles, Calif.
TERENCE CAWTHORNE, M.D., London, England.

MODERATOR SOOY: First, I will introduce Dr. Victor Goodhill, who will talk to us on the principles of tympanoplasty.

THE SURGICAL PHYSIOLOGY OF TYMPANOPLASTY.*

VICTOR GOODHILL, M.D.,

Los Angeles, Calif.

INTRODUCTION.

The term "tympanoplasty" has been applied to that group of surgical procedures in which the principles of reconstructive surgery are applied to the restoration of middle ear function.

For many decades, otologists have been aware of the necessity of such surgical reconstructions, and a number of attempts in this direction have been recorded. There are numerous instances of improvement in hearing following various forms of modified radical mastoid operations, but these occurrences were haphazard and usually not planned physiologically. No definitive methods and no techniques with predictability were available until the recent contributions of Wullstein,[1] Zollner,[2] Pietrantoni[3] and others.

*Presented as part of a Symposium on Tympanoplasty at the meeting of the Western Section, American Laryngological, Rhinological and Otological Society, Beverly Hills, Calif., March 4, 1958.

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At present, we are beginning to see the emergence of a positive plan of surgical attack with increasing predictability for restoration of tympanic function; however, we are still far from the time when results can approach the predictability of fenestration and even of stapes mobilization surgery.

It is not surprising that the emergence of this branch of otologic surgery had to wait until the maturation of the principles of plastic and reconstructive surgery and the acquisition of fairly substantial knowledge of tympanic physiology. The latter is still far from complete, but is adequate for practical application in many instances.

It is the purpose of this presentation to correlate some of the known details of middle ear physiology and possible surgical approaches to the solution of tympanic problems. Basic principles only will be presented. Neither technical details nor results will be reported in this paper.

BASIC TYMPANIC PHYSIOLOGY.

The middle ear mechanism is essentially an impedance matching mechanism, or a "transformer" to help air-borne sound travel with increased force into the liquid perilymph with no significant alterations in frequency characteristics. This is accomplished primarily by a combination of two physical mechanisms: *1.* the difference in area between the effective vibratory portion of the tympanic membrane and that of the stapedial footplate; *2.* the lever action of the ossicular chain. Contrary to some conceptions, it is primarily the area difference which is responsible for the sound pressure transformer action. In man this is approximately 22:1. The lever action of the ossicular chain is approximately 1.1:1, and the product yields a result of approximately 26 db., which is the essential contribution of the entire middle ear mechanism to the transmission of air-borne sound to the cochlear perilymph; however, there are other factors in middle ear physiology which must be considered before one can understand the application of tympanoplastic procedures.

The normal tympanic membrane is essentially transparent to sound waves, and thus the round window is not primarily

shielded from air-borne sound. An intact ossicular chain is necessary so that a significant sound pressure difference will exist between the two windows and thus between the two scalae allowing basilar membrane motion.

The Eustachian tube has an important function in maintaining essentially equal pressures on both sides of the tympanic membrane, thus avoiding painful stretching of the drum following ambient pressure changes in the external canal. This equalization allows maximum response under wide pressure changes.

It is of course possible for conductive lesions to reach db. loss values far in excess of the 26 db. described above as the contribution of the drum membrane and ossicular chain to sound pressure transformer action. In cases with intact drum but ossicular discontinuity, the drum may become worse than useless, since it may both reflect and absorb sound energy. Ankylosis of one of the windows may add another 25 to 30 db. of loss to the function of the middle ear; thus conductive lesions may reach losses of 60 db. and more. In extreme conditions, where scar tissue may simultaneously immobilize both the round window membrane and the stapedial footplate, it is conceivable that such an "acoustic short circuit" may even further increase the transmissive loss, although such a loss actually involves some perilymph immobilization, and may not truly be described as a middle ear lesion *per se*.

In addition to the above, there are problems of tympanic physiology which may involve the tympanic muscles as well as the suspensory ligaments of the ossicles. Such physiological problems will be reflected in the functional efficacy of the ossicular chain lever action, but in addition may produce increased stiffnesses and frictional problems resulting in varying deficits to acoustic transmission.

There are rather marked differences between the numerical values obtained in the human and in various experimental animals. Thus, much of the recent research data from cat and guinea pig studies are not so readily transferred to the human as had previously been considered possible. We must be alert to these major species differences in our interpreta-

tions of experimental data obtained in lower animal forms, and extrapolated for human application.

In summary, normal tympanic or middle ear physiology demands the existence of a patent external auditory canal (see Fig. 1), a normal receptor mechanism via a normally functioning tympanic membrane, continuity of the tympanic membrane and a normally functioning ossicular chain, with a mobile stapedial footplate, a normally yielding round window membrane, and finally a well regulated tympanic air space provided by normal Eustachian tube function. In the absence of a functioning ossicular chain it is necessary for the round window also to be shielded.)

TYMPANIC FUNCTION REQUISITES.

1. Patent external canal.
2. Normal drum (pick up).
3. Intact continuity of drum, ossicular chain and mobile footplate (transmission-piston).
4. Yielding but shielded round window membrane (sound pressure difference in absence of ossicular chain).
5. Tympanic air space equalization by normal tubal function.

Fig. 1.

Obviously, not all of these requirements can be satisfied in every tympanoplastic procedure, and many compromises are necessary in the application of tympanoplastic surgical principles to the solution of middle ear infections, congenital defects, and other lesions; furthermore, the evaluation of a given conductive loss requires a very careful analysis of all possible contributing factors from all regions of the tympanic mechanism.

LESIONS REQUIRING TYMPANOPLASTY—THE PATHOLOGIC STATES.

A number of conditions involving the middle ear may respond to tympanoplastic maneuvers. They may be simply divided into the three groups; *a.* congenital lesions; *b.* infections and trauma; and *c.* cholesteatoma and other ear tumors.

a. Congenital Lesions.

A number of congenital anomalies present themselves for

solution, and many of them may be solved by tympanoplastic procedures. The well recognized congenital defect of the external auditory canal, tympanic membrane and ossicular chain is best treated by a properly selected tympanoplastic procedure. It is generally not recognized, however, that there are a number of congenital defects of the middle ear which may present themselves with normal tympanic membranes. These lesions may include not only anomalies of the malleus, incus and stapes, but also congenital fixation of the footplate. They are frequently accompanied by absent stapedius and tensor tympani muscles, as well as by deformities of the round window niche and even the promontory.

TYMPANIC PHYSIOLOGIC PROBLEMS.

1. Acoustic reception lesions.
2. Acoustic transmission lesions.
3. Window fixations.
4. Loss of sound pressure difference.
5. *Acoustic* short circuits.
6. Multiple combinations of above.

Fig. 2.

b. Infections and Trauma.

The most common group of lesions requiring tympanoplastic surgery are those produced by the sequelae of otitis media in which there may occur various combinations and degrees of disruption of the tympanic membrane-ossicular chain mechanism. Similar lesions may follow both direct and indirect trauma to the ear.

c. Cholesteatoma and Other Ear Tumors.

The most common space occupying lesion of the middle ear is cholesteatoma, of which there are a number of varieties. Here again the dissolution of continuity of the middle ear mechanism is best treated by tympanoplastic surgical principles.

LESIONS REQUIRING TYMPANOPLASTY—THE FUNCTIONAL STATES.

The functional lesions that may disturb middle ear physiol-

ogy (see Fig. 2), may be divided into the following groups: *a. Acoustic reception lesions* involving the sound pick-up action of the drum membrane, and most commonly produced by perforations of varying sizes and locations. In accordance with the well-known studies of Békésy[4] and others, there are major differences in the sequelae of perforations, depending upon their size and location in relation to the manubrium and to the annulus; *b. Acoustic transmission lesions,* involving the ossicular chain. These lesions may involve any of the three ossicles in whole or in part. They may be manifested not only by loss of continuity, the most common example of which is the aseptic necrosis of the lenticular process of the incus, but also by increased stiffness states involving the ossicular joints through arthritic fixations. Occasional atrophic elasticity of incus or stapes crura may decrease transmission; *c. Fixation of windows,* especially the oval window, by ankylosis of the footplate and occasionally osteogenic closure of the round window niche and effective blocking of the round window membrane. *d. Loss of the normal difference in sound pressures delivered to the two windows.* There is normally an effective difference in force of the sound pressures reaching the two windows. Usually, of course, this occurs through the trans-ossicular excitation of the footplate in the oval window and through some minor blocking of air-borne sound to the round window by the curtain action of the tympanic membrane. In situations where there is a loss of ossicular continuity, there may be an equal sound pressure level reaching both windows, which will decrease the possibility of basilar membrane motion; *e. Acoustic short circuit* between the two windows by the effect of a scar tissue band running between the footplate and in contact with the round window membrane; *f. Multiple combinations* of any of the above lesions, and also combined with some degree of perilymph immobilization by chronic labyrinthitis.

PRE-OPERATIVE ASSESSMENT TECHNIQUES.

In one crucial respect, tympanoplastic surgery requires the same pre-requisite as fenestration and stapes mobilization surgery; namely, in the measurement and evaluation of coch-

lear reserve. It is of little value to subject a patient to a major reconstructive procedure or procedures, to attempt restoration of tympanic function in the presence of a lesion of the cochlea sufficiently great to thwart a possible significant restoration of hearing; therefore, a careful assessment of the integrity of cochlear reserve is the first necessary preoperative physiological study. This can usually be done adequately by pure tone audiometry with quantitative masking for bone conduction thresholds, speech reception thresholds and speech discrimination scores. In some instances, recruitment studies may be indicated, but usually these are not necessary. The demonstration of an adequate air bone

PRE-OPERATIVE TYMPANIC FUNCTION TESTS.

1. *Cochlear Reserve and Bone-Air Gap Tests.*
   - A. Pure tone audiometry (A. C. and B. C.).
   - B. Quantitative masking for bone conduction.
   - C. Speech reception threshold.
   - D. Speech discrimination scores.
   - E. (Recruitment tests).
2. *Prostheses Tests.*
   - A. Drum covering.
   - B. Columellization.
   - C. Blocking.
3. *Acoustic Probe Tests.*
   - A. Mechanical (Pohlman) probe.
   - B. Audiometric B. C. vibratory probe.

Fig. 3.

gap (at least 15 to 20 db.), a bone conduction threshold not lower than 45 db. and a speech discrimination score not lower than 50 per cent are necessary pre-requisites.

With demonstration of good cochlear reserve, the next function of preoperative testing is an attempt to gather qualitative, as well as quantitative, differential audiologic data regarding the status of the various middle ear components. This requires a number of techniques (see Fig. 3), which may be tailored to suit the individual case. The following are the basic procedures usually necessary in most cases: *a. The drum covering prosthesis.* This is simply a patch applied over

a perforation to increase the area of sound reception. In the presence of an intact ossicular chain with no other obstructive lesions, such a test will usually reveal a significant improvement in hearing; *b. A columellization prosthesis;* a capsule or Korojel (Pohlman) type of prosthesis applied to the incudostapedial joint, or to the capitulum of the stapes, will usually yield information regarding the status of the oval window in cases of suspected ossicular discontinuity or footplate fixation; *c. Blocking prosthesis;* a similar capsule or Korojel (Pohlman) type prosthesis, or cotton oil plug applied to the

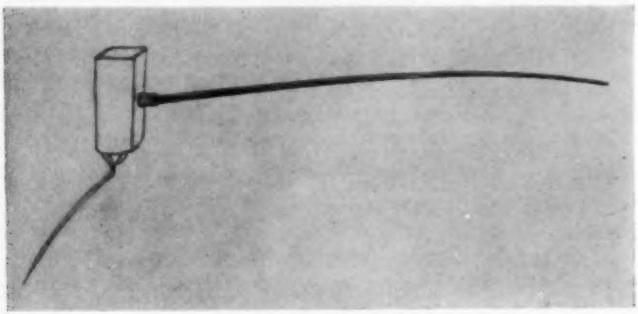

Fig. 4. Electro-Acoustic Probe.

region of the round window niche as a blocking prosthesis will give information regarding the sound pressure differences of the two windows; *d. Probe audiometry* is of tremendous value in quantitative as well as qualitative estimation of integrity of the ossicular chain and the windows. There is divided opinion as to the necessity of using probe audiometry in surgery, but its utilization diagnostically is conceded to be important by most workers in the field.

Probe audiometry has been in use for a number of years after its introduction and popularization by the late A. G. Pohlman.[5] His probe was a simple mechanical rod attached to a vibrating plastic membrane. This mechanical probe test yields valuable differential tympanic information of qualitative nature.

The electro-acoustic or audiometric B. C. probe type of test yields similar qualitative data, but also yields quantitative information for more precise comparison of tympanic responses, as illustrated in Figs. 4 to 9. At present, it is not possible to establish an accurate base line for "normal" responses, since there are major variables in the pressure of

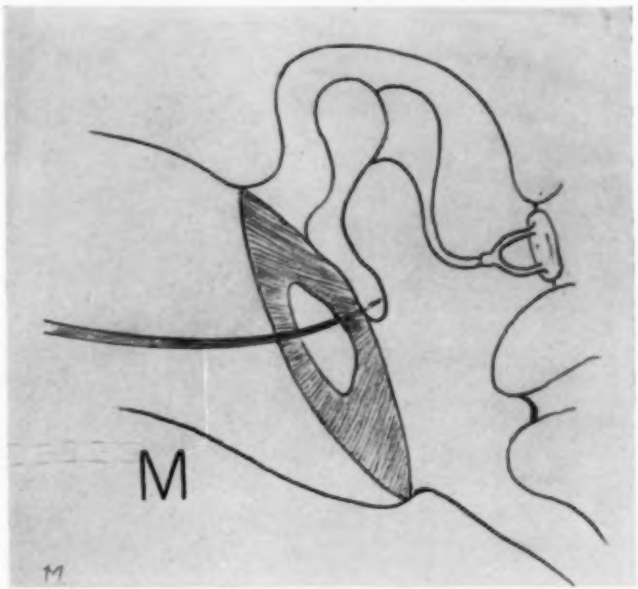

Fig. 5. Probe in Contact with Malleus.

application, size of applicator, etc.; however, the comparative data obtained are most informative, and such careful tympanic exploration will frequently be of decisive character in planning tympanoplastic approaches.

The combined evaluation of data obtained by all of the above methods is invaluable in localizing the location and extent of middle ear lesions and in planning appropriate tympanoplastic solutions for these problems.

BASIC TYMPANOPLASTIC OPERATIONS.

The basic surgical approaches for restitution of middle ear function may be described under the two heads of, *a.* Tympanolysis; and *b.* Tympanoplasty.

a. Tympanolysis.

This term applies to the surgical treatment of scars and

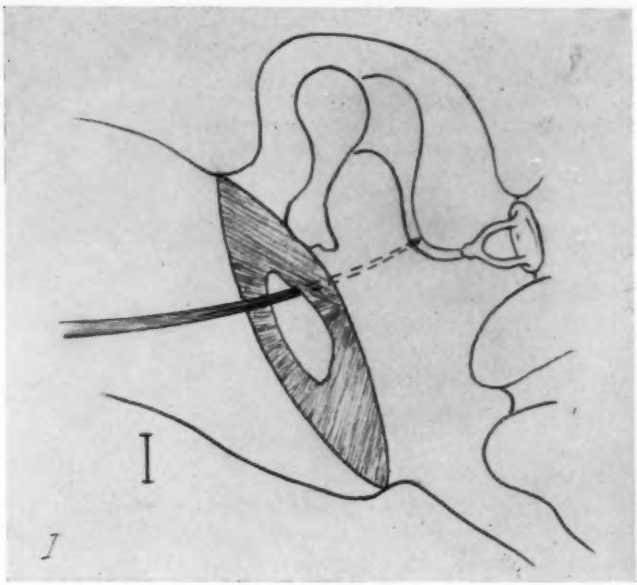

Fig. 6. Probe in Contact with Incus.

fibrous tissue masses partially or completely immobilizing an otherwise normal drum membrane and ossicular chain. (In addition, there is the necessity for removal of such scar tissue bands in many of the tympanoplastic procedures listed below). There are many cases of conductive deafness with intact drum membranes and anatomically and physiologically intact ossicular chains. Here the entire problem is that of

middle ear scarring with not only obliteration of the air space, but partial immobilization of the stapes by a "blanket" of thickened mucoperiosteum. In these cases there is justification for tympanolysis alone without any of the tympanoplastic procedures listed below.

Tympanolysis can be divided into an anterior and posterior

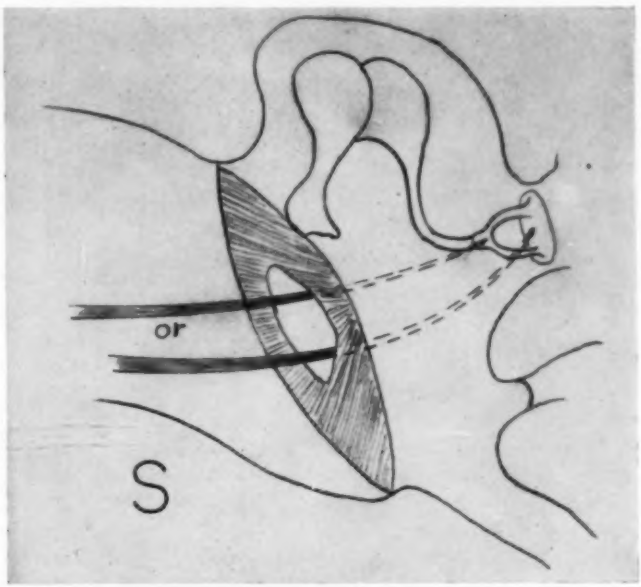

Fig. 7. Probe in Contact with Either Stapes Capitulum or Footplate.

type. In some instances it is necessary to use only the one approach, and in others both approaches are indicated.

Posterior tympanolysis is the most common type required, and can be accomplished by an exploratory tympanotomy, utilizing the typical stapes mobilization middle ear incisions in which the posterior one-third to one-half of the middle ear is adequately exposed (see Fig. 10), and scar tissue lesions can be removed (see Figs. 11, 12). In instances where clin-

ical as well as operative findings in a posterior tympanolysis indicate a lesion anteriorly, an anterior tympanolysis is then performed. This involves a triangular incision of the tympanic membrane as illustrated in Fig. 13, in which the anterior portion of the tympanic cavity is exposed, particularly in relation to the Eustachian tube orifice. After the exposure

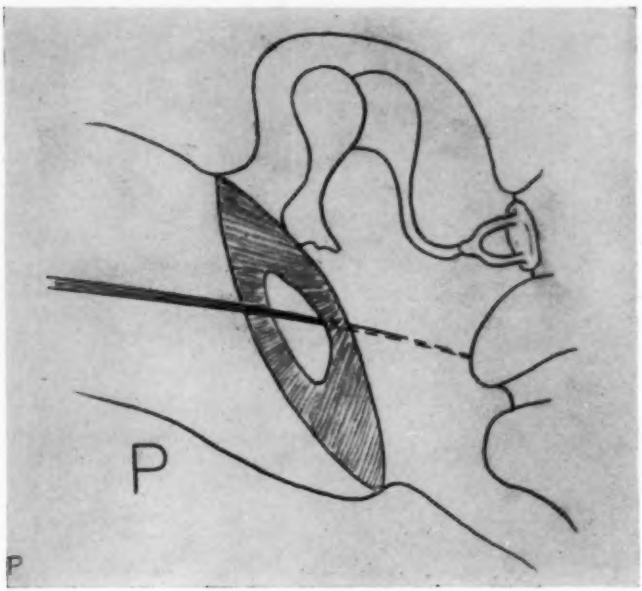

Fig. 8. Probe in Contact with Promontory.

has been made (see Fig. 14), appropriate instrumentation is performed for scar removal and the incision closed. In the closure, occasional middle ear support for the flap is necessary with one or two pieces of gelfoam. Occasionally a small skin graft is indicated to cover any anterior defect produced by retraction of fibers of the fibrous layer of the tympanic membrane.

b. Tympanoplasty.

Wullstein made an excellent contribution to progress in this branch of surgery by giving us his schema, in which he presents five types of tympanoplasty based upon anatomical and pathological findings. Since these five types have been

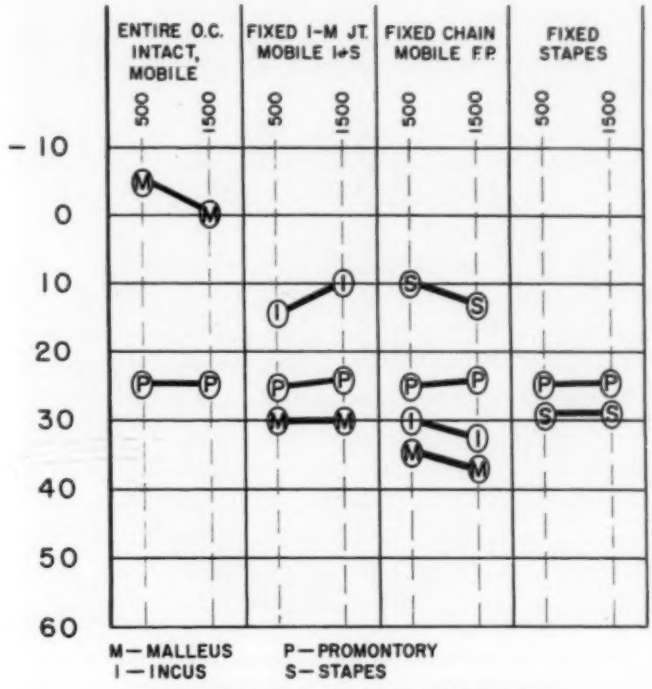

Fig. 9. Representative Probe Audiograms.

widely adopted and have simplified discussions, it appears logical to utilize them in this presentation. In the following tables and charts, the designations "Tympanoplasty Types I, II, III, IV, and V" will apply to the Wullstein classification to be given below:

Type I. (Myringoplasty).

This is the simplest problem to solve surgically in tympanoplasty. It refers to the closure of a perforation in the drum where there is an intact normally mobile ossicular chain and there are no other significant pathologic findings (see Fig. 15).

Fig. 10. Posterior Omega Incision.

Type II. (Myringoplasty).

In this more advanced version of myringoplasty, there may be absence of a portion of the malleus or incus (see Fig. 16), but there is still physiological continuity of the entire ossicular chain in spite of these defects. This may be termed an expanded myringoplasty, but basically it is the same sort of

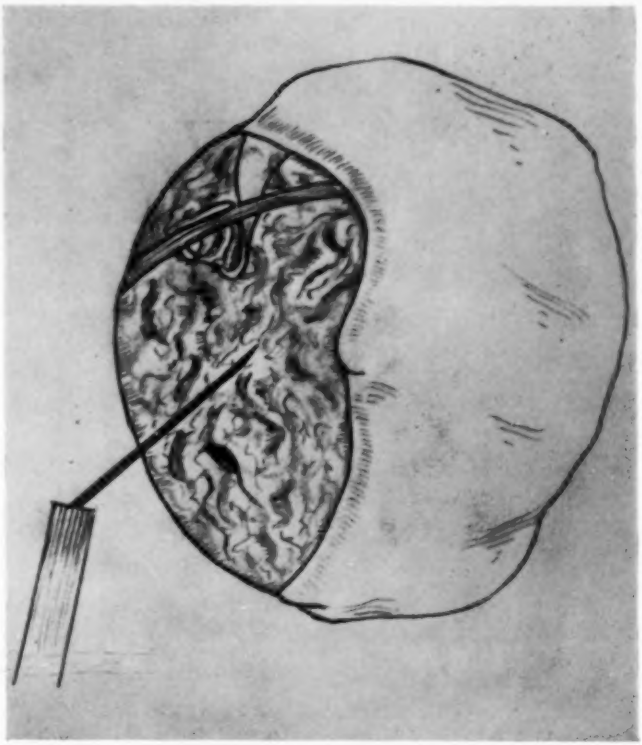

Fig. 11. Posterior Tympanolysis with Removal of Diffuse Posterior Scars.

procedure involving only the repair of the acoustic receptor, so that the area difference between the drum and the stapedial footplate may be restored, thus restoring the sound pressure transformer action.

Type III. (Columellization Operation or Myringostapediopexy).

In this very important type of procedure applied to cases where there is dissolution of continuity of function of malleus and incus, but normal anatomical and physiological integrity

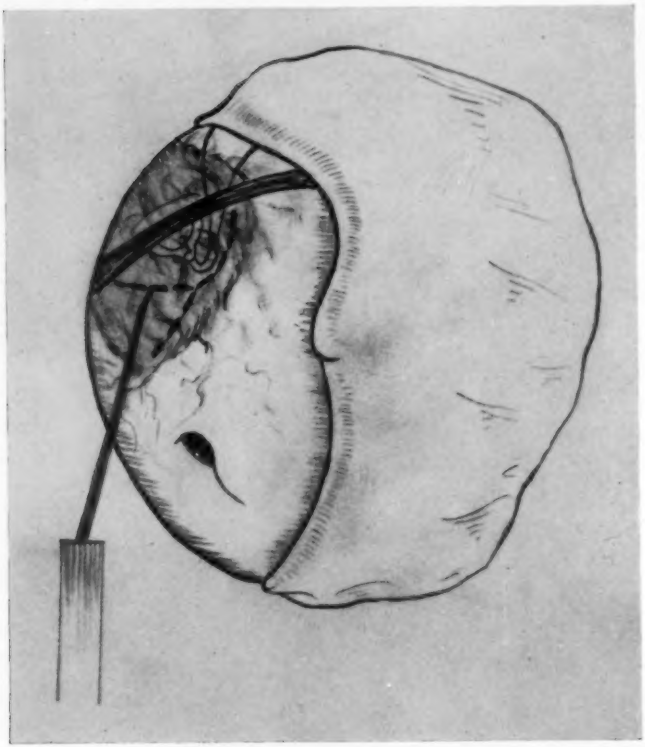

Fig. 12. Posterior Tympanolysis with Dissection of Peri-stapedial Fibrous Tent.

of the stapes and the stapes footplate, a graft is applied directly to the head of the stapes (see Fig. 17). This may either be done in cases where there is still a serviceable peripheral tympanic membrane remnant, or in instances where there is no tympanic membrane at all, as in post radical mastoidectomy cases. In the latter instance a larger and thicker graft may be necessary, but the basic principle remains the same; namely, that the tympanic membrane (either restored or recreated) is anchored directly to the head of the stapes. In this approach, the piston action of the drum to the stapes footplate

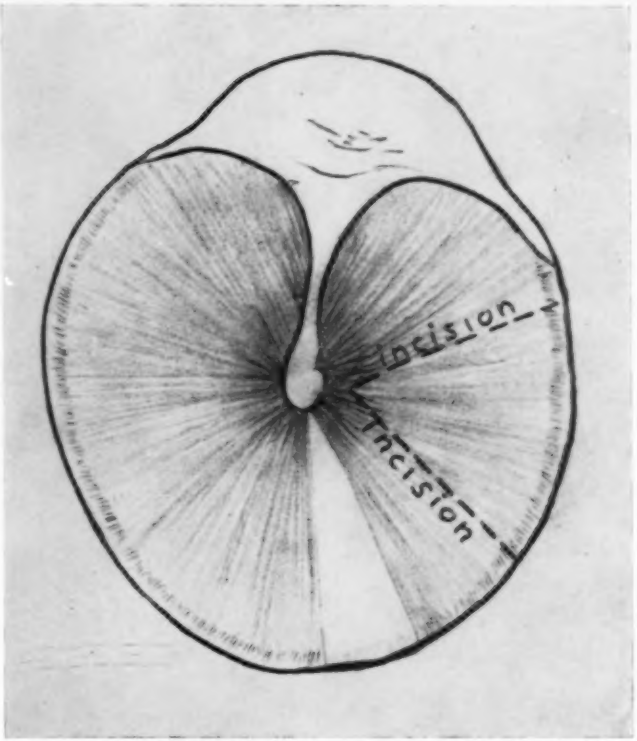

Fig. 13. Anterior Tympanolysis Incisions.

is still maintained, and the sound pressure transformation can be restored to a very efficacious degree, notwithstanding the loss of the lever action of the malleus and incus.

Type IV. (The Cavum Minor Operation or Sound Baffle Operation, or Round Window Operation).

In instances where there has been a loss of stapedial capitulum and crura, but where the footplate is still mobile, the creation of the Cavum minor, or closed hypotympanum, may yield sufficient sound protection to the round window mem-

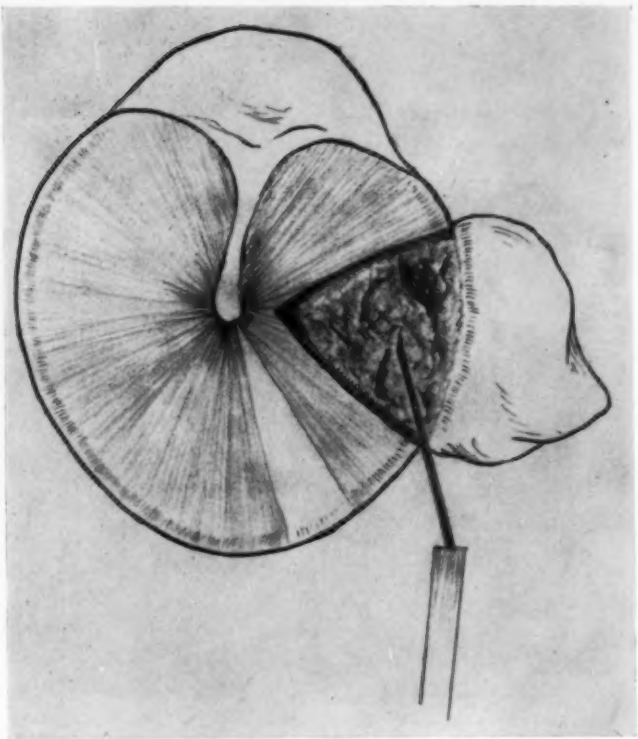

Fig. 14. Anterior Tympanolysis with Dissection of Peri-tubal Fibrous Tissue.

brane so as to restore the sound pressure difference between the two windows (see Fig. 18-A). In this procedure, a mucosal graft is rotated inferiorly from the promontory over the round window niche and covered with a skin graft in continuity with the Eustachian orifice, thus creating a small middle ear which has tubal continuity and in which the oval window region is exteriorized to air-bone sound (see Fig. 18-B). The round window acoustic baffle effect will frequently allow restoration of thresholds to the 30 db. level. The obvious loss of the transformer action of the middle ear still exists, but

the sound pressure difference may be adequate for significant gains in hearing.

Type V. The Fenestration Operation Combined with the Cavum Minor Operation.

In instances where there is a fixed stapedial footplate there may still be hope for restoration of hearing by an adequate

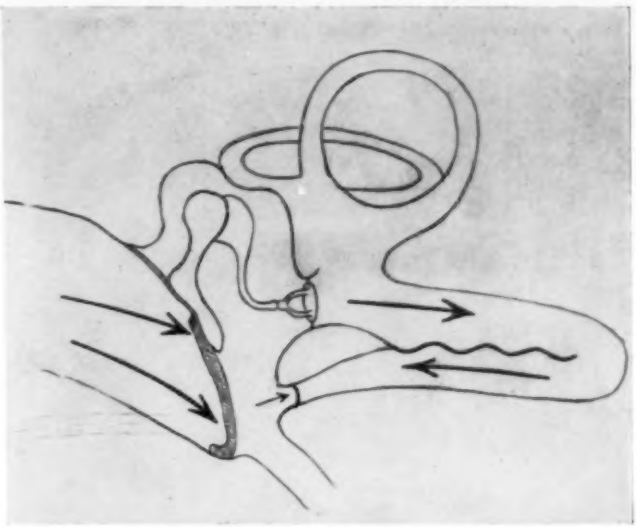

Fig. 15. Type I—Tympanoplasty (Myringoplasty). Note normal ossicular chain with graft covering drum perforation.

round window baffle procedure as described above, combined with fenestration of the horizontal semicircular canal, again yielding a level of possibly 30 db. or better in some instances (see Fig. 19).

(In certain instances where it is possible to mobilize the footplate with ease, one may combine a stapes mobilization with a Type IV, instead of resorting to a fenestration.)

The approach to Types I, II, III, and IV tympanoplasty may

be either transmeatal or transmastoid. The transmastoid approach varies quite a bit as to route of entry; thus the post-auricular, supra-auricular, and endaural approaches are all in use at the present time. Type V tympanoplasty cannot be done transmeatally, since it involves a fenestration and must always be done through a transmastoid approach.

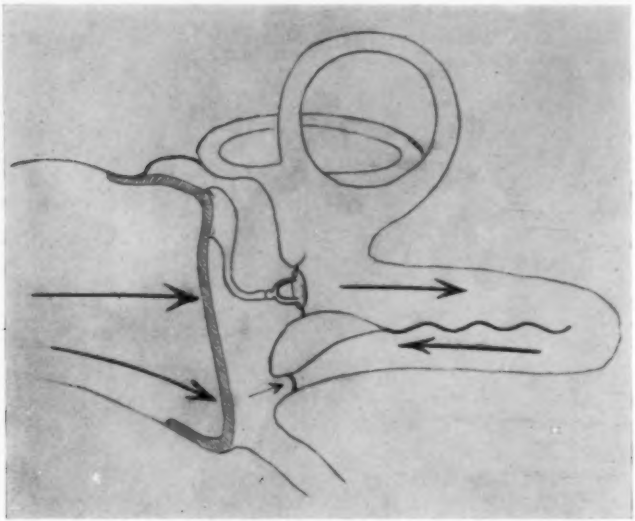

Fig. 16. Type II—Tympanoplasty (Expanded Myringoplasty). Note absence of malleus with skin graft covering perforation and in contact with incus.

SURGICAL AUDIOMETRY.

At the present time there is no uniformity of practice with regard to surgical audiometry in tympanoplasty. Some otologists prefer to do this type of surgery under local anesthesia with great reliance upon surgical audiometry, while others feel that the audiometric information obtained pre-operatively is adequate, and that the observations under the dissecting microscope fulfill the necessary criteria for decisions and, therefore, they do their procedures under general anesthesia.

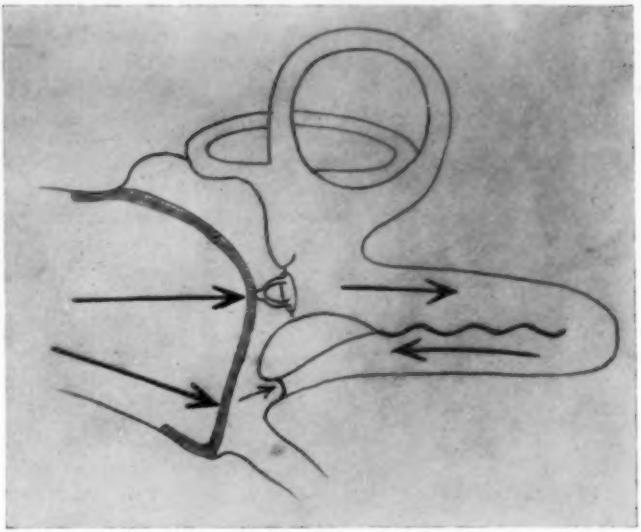

Fig. 17. Type III—Tympanoplasty (Columellization or Myringostapediopexy). Note absence of malleus and incus. Graft covers perforation and is in contact with stapedial capitulum.

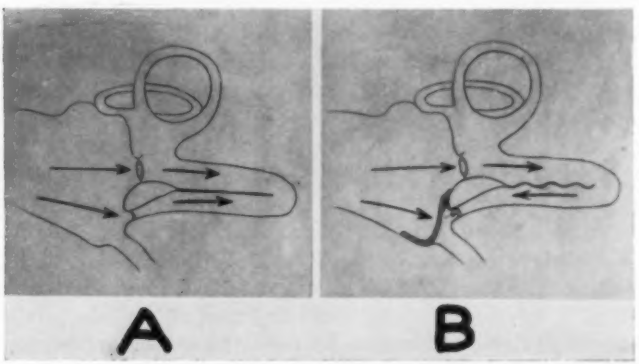

Fig. 18. Type IV—Tympanoplasty (Cavum Minor or Sound Baffle or Round Window Operation). A. Note loss of stapedial crura, but mobile footplate. Nevertheless, because of lack of sound pressure difference, the basilar membrane does not move. B. Creation of cavum minor blocks the round window membrane and re-establishes a sound pressure difference. Note basilar membrane in motion.

There probably is a place for both local and general anesthesia in this group of cases. There may very well be problems which can be solved better by surgical audiometry in some instances than in others. Further experience and reports of many observers will help to answer this question.

Of the methods available for surgical audiometry, two seem

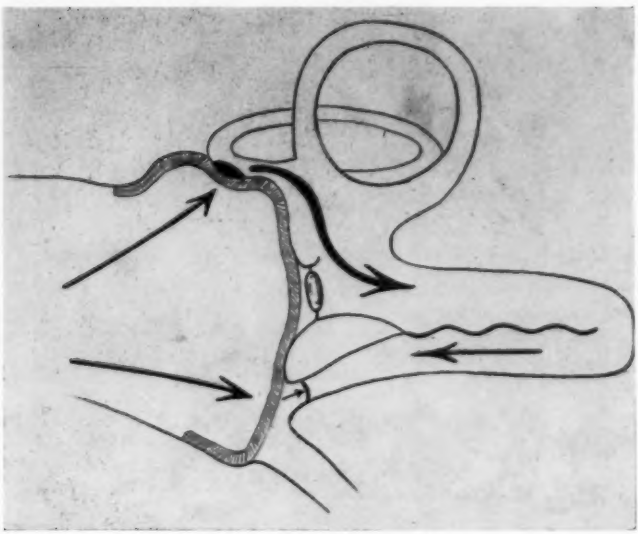

Fig. 19. Type V—Tympanoplasty (Fenestration Combined with Cavum Minor). Note sound protection of round window and new entrance for sound through semicircular canal fenestra.

to be of greatest importance: *a.* the utilization of acoustic probe audiometry as described previously and as illustrated in Fig. 9, in which definitive information of various portions of the ossicular chain may be obtained; and *b.* surgical audiometry utilizing a modified nomograph, as described by the author and his colleagues in stapes mobilization surgery. The latter may be applied to tympanoplastic surgery in certain instances, since the basic principles of closure of the air-bone

gap apply just as forcefully to tympanoplasty as they do to stapes mobilization surgery.

REPRESENTATIVE MIDDLE EAR PROBLEMS AND THEIR TYMPANOPLASTIC SOLUTIONS.

Scores of pathologic possibilities may present themselves to the otologist for solution. In an attempt to classify some of these problems, the following table (see Fig. 20), was devised. It lists a number of possible combinations of lesions, all of which have been encountered by the writer. It must be remembered that all of these conditions may exist, either alone or in combination with some degree of mastoid disease. Accordingly, the solution of these problems may be approached either through tympanotomy alone or through a combination of tympanotomy and mastoidectomy. Thus, almost 80 possibilities may be considered in this tabular presentation. Actually, the number of possibilities is infinitely greater when one considers the various combinations and cross combinations that may be encountered.

EVALUATION OF RESULTS.

It is not the purpose of this presentation to discuss results, except to point out that the evaluation of surgical results in this surgery should be no different from the method of evaluating the results of any type of middle ear surgery, including stapes mobilization and fenestration surgery. As the author and his associates have pointed out in the past, and as has been pointed out by others, the only valid method of assessing *efficacy of surgical technique* in middle ear surgery is in the measurement of the degree of closure of the air-bone gap. This evaluation technique must be considered, notwithstanding the practical aspect of the restoration of hearing to economic and socially desirable levels, (usually the 30 db. level). We are all agreed that the latter is an extremely important final goal; however, when we are in the early stages of development of a branch of middle ear surgery, and are anxious to evaluate varying techniques and approaches, no method of evaluation other than that involving the degree of closure of the air-bone gap can yield any valid data. It

	Ossicles Normal and Normal Function of Ossicular Chain	Minor Malleus or Incus Lesions but Normal Function of Ossicular Chain	Defective Ossicular Chain but Normal Stapes	Defective Stapes but Normal Function of Footplate	Fixed Stapes Footplate
M. T. intact and scarred * * * No tymp. fibrosis * * * Open E. tube	0-10 db. No Rx.	0-10 db. No Rx.	30-50 db. T. plasty III	30-50 db. T. plasty IV	45-60 db. Stapedolysis (P. B.) + T. plasty IV OR T. plasty V.
M. T. intact and scarred * * * Tymp. fibrosis * * * Open E. tube	15-35 db. Post. T. lysis	15-35 db. Post. T. lysis	35-65 db. Post. T. lysis + T. plasty III	35-65 db. Post. T. lysis + T. plasty IV	45-65 db. Post. T. lysis + Stapedolysis (P. B.) + T. plasty IV OR T. plasty V.
M. T. intact and scarred * * * Tymp. fibrosis * * * Closed E. tube	15-40 db. P. & A. T. lysis	15-45 db. P. & A. T. lysis	35-65 db. P. & A. T. lysis + T. plasty III	35-65 db. P. & A. T. lysis + T. plasty IV	45-65 db. P. & A. T. lysis + Stapedolysis (P. B.) + T. plasty IV OR T. plasty V.
M. T. perforation * * * No tymp. fibrosis * * * Open E. tube	15-25 db. T. plasty I	15-25 db. T. plasty II	30-55 db. T. plasty III	30-60 db. T. plasty IV	35-60 db. Stapedolysis (P. B.) + T. plasty IV OR T. plasty V.

M. T. perforation * * * Tymp. fibrosis * * * Open E. tube	20-40 db. Post. T. lysis + T. plasty I	20-40 db. Post. T. lysis + T. plasty II	30-60 db. Post. T. lysis + T. plasty III	30-60 db. Post. T. lysis + T. plasty IV	40-60 db. Stapedolysis (P. B.) + Post. T. lysis + T. plasty IV OR T. plasty V.
M. T. perforation * * * Tymp. fibrosis * * * Closed E. tube	25-45 db. P. & A. T. lysis + T. plasty I	25-45 db. P. & A. T. lysis + T. plasty II	30-60 db. P. & A. T. lysis + T. plasty III	30-60 db. P. & A. T. lysis + T. plasty IV	40-60 db. Stapedolysis (P. B.) + P. & A. T. lysis + T. plasty IV OR T. plasty V.
No M. T. * * * No tymp. fibrosis * * * Open E. tube	Unlikely	Unlikely	30-55 db. T. plasty III	30-55 db. T. plasty IV	40-60 db. Stapedolysis (P. B.) + T. plasty IV OR T. plasty V.
No M. T. * * * Tymp. fibrosis * * * Open E. tube	Unlikely	Unlikely	30-55 db. Post. T. lysis + T. plasty III	30-55 db. Post. T. lysis + T. plasty IV	40-60 db. Stapedolysis (P. B.) + Post. T. lysis + T. plasty IV OR T. plasty V.
No M. T. * * * Tymp. fibrosis * * * Closed E. tube	Unlikely	Unlikely	35-60 db. P. & A. T. lysis + T. plasty III	35-60 db. P. & A. T. lysis + T. plasty IV	40-60 db. Stapedolysis (P. B.) + P. & A. T. lysis + T. plasty IV OR T. plasty V.

Fig. 20. Table showing the possible pathologic lesions, the accompanying hearing losses, and the recommended reconstruction techniques.

Abbreviations—M. T., Membrana Tympani; Tymp., Tympanic; E. tube, Eustachian tube; T. plasty, Tympanoplasty; T. lysis, Tympanolysis; P. B., Peribasal; P. & A., Posterior and Anterior.

is the feeling of the author that the per cent improvement technique which we have previously described in stapes mobilization surgery, should also be used in the evaluation of results in tympanoplastic surgery. This per cent improvement technique is easily arrived at by dividing the decibel gain post-operatively by the air-bone gap:

$$\text{Per cent Improvement} = \frac{\text{A. C. Gain (db.).}}{\text{B. C.-A .C. Gap (db.).}}$$

SUMMARY AND CONCLUSIONS.

1. Basic physiological principles underlying tympanoplastic surgery are presented. Pre-operative and operative evaluative techniques are described, and the multiple possibilities of tympanoplastic approaches are tabulated.

2. A methodology for evaluation of results is presented involving the per cent improvement technique, based upon degree of closure of the air-bone gap.

3. Tympanoplastic surgery is in a very early stage of development. It is based largely upon the application of plastic surgical principles to recently acquired knowledge of tympanic physiology. It is interesting to speculate on the increased knowledge of tympanic physiology which will accrue as the result of tympanoplastic surgery itself. Never before has it been possible to acquire as much quantitative data under microscopic inspection conditions of the middle ear as during this present new era of tympanoplastic surgery.

4. Tympanoplastic surgery must be based upon careful evaluation of the physiologic state of the middle ear in every patient. No simplified rote operation can be used without such evaluation. It is better to think of tympanoplasty as a surgical approach than as an operation *per se*.

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MODERATOR SOOY: I am sure I speak for all of us when I say how grateful we are to Dr. Goodhill for giving us this excellent paper. The next paper has to do with the clinical experience in tympanoplasty. It will be presented by Dr. Leland House of Los Angeles.

CLINICAL EXPERIENCES IN TYMPANOPLASTY.*

Results in Sixty-One Cases.†

LELAND R. HOUSE, M.D.,

Los Angeles, Calif.

Since "Plastic Surgery" means repair or reconstruction of a part, I have chosen to include any procedure which is done to repair or reconstruct any of the tympanic structures within the subject of "tympanoplasty."

This would then include surgical repair of simple perforations of the tympanic membrane alone, or combined with any changes in the ossicles and middle ear membranes and windows for the purpose of eradicating disease, and/or preserving or restoring hearing. Various names are appropriately applied to more specific procedures, such as myringoplasty, transmeatal tympanoplasty (Bell), myringostapediopexy (Juers), tympanomalleolar stapedopexy (Bell), endaural tympanoplasty, and others.

It seems appropriate to include all of these procedures in our general thinking on the subject of tympanoplasty, but time and experience plus usage will encourage the use of more specifically identifying terms. Time will not permit elaboration on each type of the above mentioned procedures. In

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preparation for this study, I have reviewed the surgical records of some 125 cases which have been done at the White Memorial Hospital during the last three years, including 61 which I have personally been able to follow in more detail.

In general, we have followed the principles of Dr. Wullstein and have followed his classification for the purpose of this study. Many details, no doubt, have fallen short of the perfection which he has attained, because of our limited experience. As I reviewed our cases I was impressed with my change in attitude toward temporal bone disease in the last few years. In previous years the available procedures for the management of the diseases of the temporal bone resulting from infection consisted principally of the modified radical mastoidectomy and the radical mastoidectomy. The question usually was, "Do we do a radical mastoidectomy or not?"

Thanks to the influence of Drs. Juers, Baron, Zollner, Wullstein, and many others, our thinking has changed from surgery of destruction to that of restoration or preservation. As evidenced by our results, there is much progress to be made. I anticipated that our results might not justify any great sense of pride in accomplishment. I do feel encouraged, however, to find that we have helped many of these patients in a manner that would not have been accomplished by our routine procedures a few years ago. Now, instead of a cut-and-dried procedure, each case is a challenge, with its own particular problem to solve. It is approached with the idea of doing whatever seems necessary to eradicate the disease and still conserve and reconstruct the hearing functions.

I have studied 61 cases in quite detail and will review points of interest.

Anesthesia: By far the most cases were done under general endotrachial anesthesia. There is no hesitancy to use local anesthesia when desirable, especially for the less extensive procedures.

Incisions: In most all cases I prefer the endaural incision. I have not used the Heermann incision, as advocated by Zollner. I have, however, used the postauricular approach on

several cases. One case was a boy 11 years old, in whom I did the Wullstein approach on one side and the endaural on the opposite side. In my experience, postoperative care is a little easier through the meatus created by the endaural incision than by the postauricular incision. One case which developed a localized perichondritis of the auricle around the tragus and antitragus occurred with the postauricular incision. In some cases a limited procedure may be accomplished through the normal meatus without an external incision.

Surgical Attack: If pathology is obviously limited to the tympanic membrane, I do not elevate the membrane to inspect the middle ear nor open the antrum or attic. In most cases, however, I am now following the plan of opening the mastoid antrum and the attic. Complete removal of mastoid cells, or the removal of disease from around the ossicles, is done according to what pathology is found. The posterior superior segment of the tympanic membrane is elevated from the annulus in most cases for inspection of the middle ear. Continuity and mobility of the ossicular chain and middle ear windows is studied. An effort is made to remove diseased polypoid mucosa, granulation tissue and cholesteatoma. If this can be done without interrupting the intact ossicular chain the ossicles will be left. The entire pars flaccida and the pars tensa may be removed without breaking the ossicular chain if indicated.

If cholesteatoma or granulations completely surround the ossicles, or if disease has caused erosion, they will be removed. In several cases the ossicular continuity had been destroyed by disease with fairly good preservation of the pars tensa. In these cases the incus was removed, the head of the malleus was removed, and the remaining drum membrane was used to seal off the tympanic cavity below the oval window creating a small tympanum.

Skin Grafts: Grafts are used to replace or cover areas where the tympanic membrane has been damaged or removed. The usual source is a free, full thickness graft from the postauricular area. Tympanomeatal flaps or flaps from the canal

wall may be used occasionally for small defects. Before applying the graft, all epithelium is removed from the recipient area to avoid burying epithelium. The promontory will be denuded in some cases to provide better source of nutrition for the graft. If the mastoid cavity is large, I prefer not to line the entire cavity with skin, but to allow it to granulate in and epithelize itself. So far I have not used delayed grafting.

To maintain separation of the graft from the middle ear mucosa, and to prevent obliteration of space, very small pieces of soaked gelfoam have been used in the hypotympanum, the Eustachian tube area and the round and oval window areas.

TABLE I.

TYPE I—13 Cases.

Healing—	
Satisfactory	8
Perforations	5
Hearing—	
Incomplete Data	2
Poor Hearing	2
Good Hearing	9

The physiologic demands necessary for preserving and restoring hearing have been well presented in former literature and reviewed by Dr. Goodhill. These requirements must be continually in mind when approaching each case.

For the purpose of discussing this series I will briefly summarize our cases from each group. Two factors were considered; the type of healing result and the comparison of preoperative and postoperative hearing.

Type I, as classified by Wullstein, includes those cases which have an intact and functioning ossicular chain and round window with some defect in the tympanic membrane. In these cases a successful repair will result in an essentially normal ear.

In this group, Type I, I reviewed 13 cases. Eight of these

cases resulted in good healing with intact tympanic membranes, and five had a recurrence with a secondary perforation in the graft. Nine had good hearing (above 30 db.), two had poor hearing, and two had no record of postoperative hearing test.

In this group, which admittedly is very small, I find no correlation in the incidence of the breaking down of the graft and the size of the original perforation. Some of these had a repair of a limited localized perforation and others had the entire tympanic membrane replaced by a graft.

Type II in this classification include those which have a slight defect involving the ossicles as well as the tympanic membrane. The ossicular defect is such that the ossicular

TABLE II.

TYPE II—22 Cases.

Healing—	
Satisfactory	16
Perforations	6
Hearing—	
Incomplete Data	4
Not Improved	6
Slightly Improved	4
Good Hearing	8

chain can still be considered functional in the repair. I have included those cases in which granulations, cholesteatoma, or fibrous tissue was found in the attic or tympanum, but still could be removed without a major dislocation of the ossicles.

In this group I reviewed 22 cases. Sixteen of these had satisfactorily healed cavities. This means that the graft is intact and apparently viable, and that the mastoid cavity is dry or free from suppuration with no more than a modest amount of crusting and debris. Six developed secondary perforations in the graft; one of these was during an episode of re-infection after an intact membrane had remained healed for 18 months. The audiometric data was incomplete in four cases, leaving 18 cases for hearing comparison. Eight cases had good hearing (above 30 db. average), and ten had poor

hearing. Of those with poor hearing, four showed definite improvement, but were still below the 30 db. level.

There were three significant complications. One was followed by a dead labyrinth. A very large cholesteatoma had eroded a segment of both the posterior and horizontal canals. On removing the mass of cholesteatoma, some of the endolymphatic labyrinth was pulled away. About ten days after surgery this same patient developed a perichondritis. Fortunately, this remained localized relatively well in the lower portion of the meatus near the tragus and antitragus, but was slow to clear. The organism was pseudomonas which, of course, is resistant to most antibiotics, and in my experience is a trouble maker. The postauricular approach had been used on this patient, but may not have had any bearing on this complication.

Another patient developed a facial paralysis which at first was partial. During the first week it became complete, but started to recover after the third week. Recovery was slow, but was complete after 16 weeks.

Type III cases are those in which there is a major defect in the ossicular chain requiring the removal of the incus and malleus, but preserving the stapes. This leaves the capitulum protruding slightly above the level of the margins of the niche of the oval window. The tympanic membrane or graft then is laid over the capitulum in contact with it. The newly created tympanic membrane then lends sound pressure transmission directly to the stapes, this being spoken of as the columellar effect. Because the graft lies close to the promontory and the pneumatic space of the middle ear is thereby made shallow, it is much easier for adhesions to form and obliterate the space.

I reviewed 10 of these cases. In seven the cavity was considered to have healed satisfactorily, in two small perforations recurred, and in one there was active secretion from an unhealed mastoid cavity. Three cases had good hearing (above 30 decibel level). Four cases had distinct and useful improvement in hearing, but still below the 30 decibel level. In

two there was no improvement, and the hearing remained at the preoperative level below 30 db.

From a theoretical estimate there should have been a better percentage of good hearing cases in this group. As near as I can determine, the chief cause for poor hearing was the reformation of adhesive processes with fixation of the flap and obliteration of the tympanic space. This impaired the freedom of one or both of the two windows.

Type IV are those cases in which the ossicular chain is entirely lost except for a mobile stapes footplate. The ideal repair in these cases seems to be to create a small tympanum

TABLE III.

TYPE III—10 Cases.

Healing—	
Satisfactory	7
Perforation or Drainage	3
Hearing—	
Incomplete Data	1
Not Improved	2
Slightly Improved	4
Good Hearing	3

with a pneumatic space which communicates with the Eustachian tube and the round window. The oval window niche is not covered at all with the graft, and is left open to receive air-transmitted sound pressure directly. The round window is protected from direct sound pressure by the tympanic membrane. This provides the phase differential that is necessary in hearing.

The small tympanum may be made ideally by the intact pars tensa if it has not been destroyed by disease. In six cases I found this possible. The ossicles were all destroyed, and the attic was diseased; but the pars tensa was sufficiently intact to use it to seal off the tympanic cavity just below the margin of the niche of the oval window. If the pars tensa is inadequte to create this small tympanum, then a skin graft is applied in one piece to cover all of the tympanic area and extend upward to cover the attic. A small button hole is cut

out of the graft corresponding to the oval window, leaving it uncovered. Several cases in this group have given surprising results with hearing in the speech range averaging 18 to 22 db.

The records of 16 patients of this type were studied. In 15 cases the cavity and graft were considered to have healed satisfactorily and one developed a secondary perforation from re-infection. Ten cases (62 per cent) had good hearing (above 30 db.), two showed improvement, but below 30 db., and three showed poor hearing with no improvement.

TABLE IV.

TYPE IV—16 Cases.

Healing—	
Satisfactory	15
Perforation	1
Hearing—	
Incomplete Data	1
Not Improved	3
Slightly Improved	2
Good Hearing	10

Percentage wise this group gave the best results of all groups, and Group III gave the worst. Factors favoring Group IV are:

First, only a small pneumatic space is needed, and this is in the region where the tympanic cavity is the deepest and most easy to preserve.

Second, the short length of skin graft bridging from one point of contact to the next makes the nutrition of the graft less critical.

Third, in these cases the greatest damage by the disease was in the attic with a relatively less severe change in the hypotympanum. The skin graft is less likely to adhere to a normal mucous membrane surface.

Wullstein's Type V are those which have a fixed stapes footplate. In these cases a stapes mobilization or a fenestration must be combined with other procedures as indicated. In

this series one case with a fixed stapes footplate was included in my Group III above. A mobilization of the footplate was done and the graft placed across the capitulum. Hearing result was not good; further surgery is contemplated.

I will summarize our findings in these 61 cases by combining the results of all of the types presented.

TABLE V.

SUMMARY—61 Cases.

Healing—		
Satisfactory	46	75%
Perforations or Discharge	15	25%
Data Incomplete	8	
Hearing—		
		% of 53
Not Improved	13	25%
Slightly Improved	10	19%
Good Hearing	30	56%

Forty-six (or 75.4 per cent) were considered to have satisfactory healing with no perforations. Fifteen had perforations or active discharge. In eight the audiometric data was incomplete, leaving 53 cases for preoperative and postoperative hearing comparison. Of the 53 cases, 30 (56.6 per cent) showed a hearing level above the 30 db. loss in the speech range. Eleven were improved but below 30 db. Twelve were not improved.

Admittedly, this is a relatively small series of cases and does not justify any fixed long range conclusions. It does lend encouragement for further efforts in this field. Time and experience may never solve all of the problems we face in this work. It does seem that another new field of otology has been opened up and that many advances may be expected in the future.

I am indebted to others on my staff who have allowed me to review their cases and assisted by giving me clinical and audiometric data. Dr. Emery Pick, who was the first to bring Wullstein's and Zollner's work to this city, has been especially helpful.

MODERATOR SOOY: Thank you, Dr. House. We will now have Dr. Cawthorne's contribution to this Symposium.

Summary and Remarks.

TERENCE CAWTHORNE, M.D. (London, England): I still do not quite know why I was called on to summarize this discussion, because my experience and knowledge of tympanoplasty is very slight; however, I have learned a great deal from the discussion this morning.

I think we all realize what a great debt we and the whole world owe to Prof. Zollner and also to Prof. Wullstein for their original work in this field, and for arousing our interest in the restoration of function where it was always assumed that it was not possible to restore function.

Dr. Goodhill mentioned the importance of the differential sound pressure, and we are indebted to Dr. Wever and to Dr. Lawrence for the clear explanation of this, which appears in their book "Physiological Acoustics."

Dr. Goodhill also mentioned ankylosis of the stapes. Now I cannot resist saying that it is not very often that the stapes becomes ankylosed in the true sense of the word. "Ankylosis" and "fixation" as applied to a joint mean that the joint is fixed, that it won't move; I do not believe that in otosclerosis the stapes is often so fixed. If it were, little or no sound would get through and the patient would be very deaf, indeed. I think that usually it is impeded. When it is really fixed, and I have under the microscope seen the stapes buried up to the neck in otosclerotic bone, that patient has had a 95 decibel loss in hearing. Such a hearing loss does not necessarily indicate poor cochlear reserve. I think that it is probably one of the physical effects of complete ankylosis of one or both windows, and that cochlear function may be unimpaired.

With regard to the question of approach, I have always thought that one should be familiar with any of the three approaches to the middle ear: posterior, endaural or endomeatral. Only then is one really in the position to be able to choose the approach most suited to the operation.

Though it has not been mentioned I do not doubt that our speakers advise using the Zeiss microscope for tympanoplasty. Wullstein, himself, makes the point that his work consists of two things: first, microscopic control in the eradication of infection; second, the restoration of function.

Anyone who has seen Prof. Wullstein will realize that tympanoplasty is not a set operation. What is done depends upon what is found, and an operation may take several hours. This is necessary in order that all infectious material is removed from the tympanic space before it is closed in by skin.

I have always felt that the danger in tympanoplasty comes from burying sepsis under skin. Wullstein and Zollner are fully aware of the danger of this, but I know from my own experience and from what I have seen of others that not everybody is so careful and painstaking in eradicating every little bit of pathology so that nothing is left behind. One of my friends who is very keen on this work and whose opinion I value highly, said that for every ten cases in which he hoped to end up with a Wullstein Type II operation—that is to say, leaving the ossicular chain more or less intact—this happened only in three. In the remainder, he had to remove the malleus and incus in order to eradicate the infection. After what we have heard from Dr. House today, it looks as though it might be a good idea to clip off the crura of the stapes as well.

In stressing the importance of not burying sepsis under skin, I have in mind that our first duty to a patient with a chronic suppurative otitis media is to make that ear safe. If we sacrifice that principle in the hope of saving or restoring hearing—laudable though the idea may be—then we are not doing our duty to the patient. Infection and its ever present danger of spread intracranially, must be eradicated before starting to save or restore hearing.

When it comes to the question of the skin graft, the speakers this morning, like Wullstein and Zollner, use full thickness skin taken from behind the ear. Some workers are claiming good results when using split thickness grafts which they find are less irritating, and are more likely to survive than whole skin. It is, however, maintained that the whole skin

gives a better chance of maintaining an air space in the lower part of the tympanic cavity.

Now this is very important, because for good hearing it is necessary to have an air space including the round window and the Eustachian tube but separated from the oval window. Both Zollner and Wullstein lay great stress on this, and they start inflating the Eustachian tube soon after the operation.

There is one other question which has not been mentioned yet which is of interest. Should tympanoplasty be advised for unilateral ear disease or should it be reserved for bilateral cases? Probably when one is beginning it would be quite a good plan to start on unilateral cases, because failure to improve hearing would not be so disappointing for the patient.

I have one final word about the Type I type of case, myringoplasty. One of my colleagues, Stuart Mawson, has been doing quite a number of myringoplasties and has been getting remarkably good results. He works through an endomeatral incision on ears with large perforations. The ear must be dry and there must be no suggestion of any chronic disease within the attic. The patient is laid up for a day or two at the most. The perforation is covered with full thickness skin, and in a very large proportion of the cases, the perforation is permanently closed.

Besides improving hearing it usually prevents the recurrent bouts of otitis media, which with large perforations so often follow on moisture getting into the ear. This avoids having to take special care when swimming, having a shower or a hair shampoo.

Finally, I was very interested to note that in the series just quoted by Dr. Goodhill and his colleagues, their best functional results followed either myringoplasty or where the only part of the ossicular chain left as the footplate of the stapes (Wullstein Type I and Type IV); thus if the surgeon can satisfy himself that he is dealing only with a case of recurrent otitis media with a large central perforation, myringoplasty is a safe and functionally satisfactory operation. Where the tympanic sepsis is persistent, and particularly when there is evidence of bone disease or erosion in the shape of foul dis-

charge granulations or cholesteatoma, a good functional result is not necessarily sacrificed by a thorough clearance of the middle ear including most of the ossicular chain.

PROGRAM OF THE SEVENTH INTERNATIONAL CONGRESS OF BRONCHOESOPHAGOLOGY.

Meeting Place—Kyoto University, Kyoto, Japan.

Friday, September 12th, 1958

8:00 P.M.-10:00 P.M.—Reception (Party) Shimomura House in Kyoto.

Saturday, September 13th

8:00 A.M.—Inaugural Session (in Kyoto University Hall).

9:00 A.M.-12:00 M.—First Scientific Session.

12:00 M.-2:00 P.M.—Luncheon (Reception. The Place not decided).

2:00 P.M.-6:00 P.M.—Sightseeing in Kyoto.

8:00 P.M.—Banquet, Miyako Hotel.

Sunday, September 14th

8:00 A.M.-11:30 A.M.—Second Scientific Session.

11:30 A.M.-12:00 M.—Closing Ceremony.

12:00 M.—Departure for sightseeing in Nara.

1:00 P.M.—Luncheon, Nara Hotel.

2:00 P.M.-6:00 P.M.—Sightseeing in Nara.

7:00 P.M.—Return to Kyoto.

THE MANAGEMENT OF HYPERKERATOSIS OF THE LARYNX.*†

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The management of hyperkeratosis of the larynx is fraught with difficulty because some of these lesions are considered precancerous, and the problems involved may defy even the most experienced laryngologist.

We know that hyperkeratotic areas in the larynx may slowly and without changes apparent to the eye, develop definite malignant characteristics. Since these changes frequently are not recognizable by visual examination, we must depend upon the microscopic diagnosis; second, we must consider the hyperkeratotic lesion as a malignant one, always aware of the fact that at any time the metamorphosis into cancer may take place.

Hyperkeratosis of the larynx appears as a thickening of the layers of the epithelium, especially the cornified portion. When it is seen as slightly raised, rather dense and thickened, white superficial plaques with evidence of inflammation, it is sometimes called leukoplakia. Hyperkeratosis, also referred to as keratosis, may be localized or diffuse in the larynx. It may occur on one cord as a grayish white, sessile growth or it may be seen occasionally as a red thickening on the cord. It may appear as a white, warty growth with sharp, spiny protuberances. It may involve all or a portion of both vocal cords, or spread to the ventricle and/or subglottic areas. A typical diffuse type of hyperkeratosis is known as pachydermia of the larynx. This is an involvement of the interarytenoid area that extends onto both vocal cords, usually up to the

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vocal process on each side. It appears as a grayish-white, sometimes red, wrinkled dry thickening of the mucosa.

Microscopically, keratosis is recognized by thickening of the epithelium, especially in the cornified stratum. As in leukoplakia, there may be evidence of chronic inflammation in the other mucosal layers or in the sub-mucosa. This is recognized by the presence of lymphocytes and plasma cells; however, the most important factor in determining the malignant tendency of a growth is the presence of cell atypism. In the most serious type of hyperkeratosis there is proliferation of the prickle-cell layer, and also the basal layer of cells, but the basal layer in the true keratotic lesion will remain unbroken. The epithelium may send projections into the subepithelial tissue, but the cells of the basal layer remain intact.

It is felt that those showing evidence of chronic inflammation and those showing involvement of the deeper layers of epithelium are more likely to metamorphose into malignant growths. At any rate the presence of atypical cells in the epithelium signifies beginning carcinomatous changes, and if these are pronounced enough the diagnosis is "carcinoma *in-situ*." When the microscopic picture shows infiltration into the subepithelium tissue, that is, the basal layer of cells are broken through by groups of carcinomatous cells, we have a true invasive carcinoma.

Since the appearance of a lesion cannot always help us in determining the presence of malignancy we must depend upon the biopsy; but frequently a biopsy may be taken from an area that does not yet show the malignant changes. The entire lesion, if it is a large one, cannot be examined microscopically, and the malignant changes may take place over a long period and at the time of the biopsy, none of these changes as yet have occurred.

We must, therefore, approach the problem with constant awareness of the danger of complacency and treat a keratotic lesion with atypical cells as though it were malignant, and yet we must not be too radical in all cases of hyperkeratosis. We come to the conclusion, then, that cases must be considered individually, according to the site of involvement, the extent,

size and appearance of the lesion as well as the microscopic findings. It is hard to formulate hard and fast rules, but we hope to indicate what we feel is best in certain typical cases.

On examination of an early hyperkeratotic lesion, one would see a small white, elevated, thickened patch on a vocal cord, perhaps several plaques on both vocal cords. They are usually seen on the upper surface of the cord, perhaps on its edge.

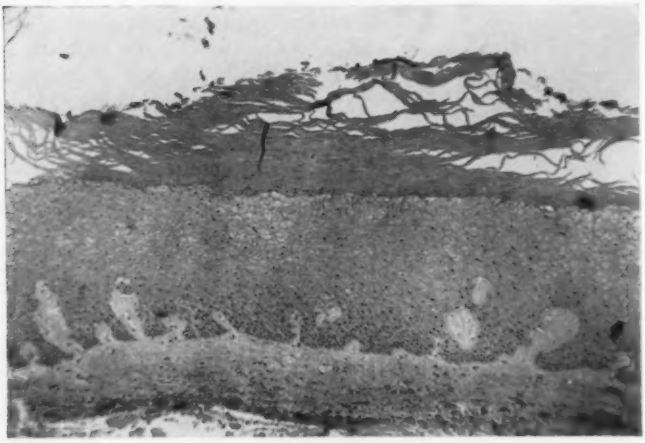

Fig. 1. Hyperkeratosis: Marked thickening of the corium; moderate thickening of the deeper epithelial layers; no significant cell atypism; no inflammatory reaction in the underlying connective tissue.

It is felt that hyperkeratotic lesions in general are due to chronic irritation. Such irritation may be caused by excessive smoking, by excessive use of alcohol, by abuse of the voice, by inhalation of fumes and dust; they may also be caused by repeated upper respiratory infections brought on by chronic infection in the sinuses and pharynx (see Case 3). If these etiological factors can be eliminated then the hyperkeratotic areas may subside or at least not increase in size; so that this is the first advice that is given, and the patient is kept under close observation. If this type of lesion does not disappear it is removed by direct laryngoscopy.

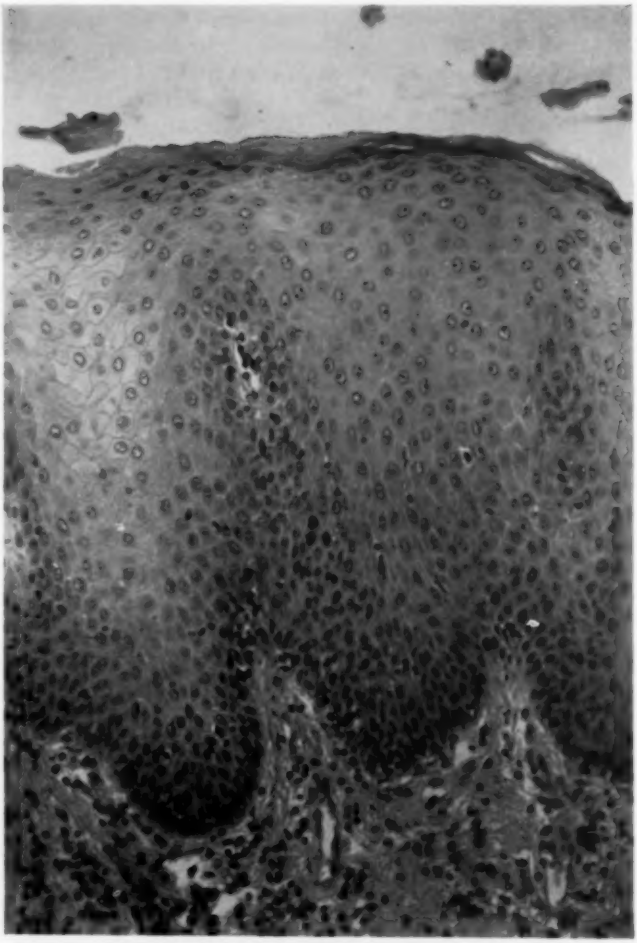

Fig. 2. Leukoplakia: Thickening of the corium, not marked; thickening of the deeper epithelial layers, more marked than on Fig. 1; adjacent connective tissue contains lymphocytes; some cell atypism (dark staining in basal layer).

The keratosis may appear as localized unilateral or bilateral thickenings. The lesion may present itself as a grayish-white area on the cord that shows evidence of chronic inflammation. The hyperkeratosis may take the form of white, warty, brittle appearing excrescences on one or on both cords. Other keratotic lesions appear as whitish or red papillary growths. It is felt that the presence of inflammation in or around the lesion or the warty, papillomatous growths are more likely to become malignant and yet the sessile, red thickening of the vocal cord may on occasion be reported as "carcinoma *in-situ*." Some of the above mentioned growths may subside under the regimen of elimination of the causative factors already indicated (see Case 2), but usually it becomes necessary to remove the keratotic lesion by biting off the involved areas or stripping the cords under direct laryngoscopy (see Case 1). Careful and frequent observation is most important, since at any time the lesions may recur (see Case 4). If they do, more radical therapy is indicated, since we know we are dealing with a growth that may become malignant.

Perhaps we would attempt several removals of the growth by direct laryngoscopy, but we will note that with each recurrence the interval is shorter, the growth seems to grow faster (see Case 8). It is best, then, to remove the lesion by doing a laryngofissure operation. For those accustomed to the technique, there may be cases with lesions small enough to be removed by suspension laryngoscopy, as suggested by Dr. LeJeune. Perhaps in the case of the lesion on an inflamed cord and the warty, papillomatous lesion we would be more inclined to do a larynofissure without attempts at stripping the cord. Clinical experience and judgment on the part of the laryngologist will dictate the procedure to be followed (see Cases 5, 7). Of course, if at any time the microscopic findings indicate the presence or the suspicion of malignancy, the growth is treated as one usually treats a carcinoma. There are some hyperkeratotic lesions that spread either to the subglottic area or to the ventricle. These should also be removed by laryngofissure (see Case 6).

So far it does not seem complicated, but when we have a keratotic lesion recurring on both vocal cords, perhaps ex-

Fig. 3. Carcinoma **in situ**: As in Fig. 2, but the basal layer of cells are quite atypical and show the characteristics of carcinoma, i.e., large nuclei, atypical chromatin arrangement; large nuceoli, hyperchromatism; large atypical shaped differentiation of squamous cells.

tending to the ventricle or the subglottic area, the situation is more seriuos. We hesitate to do a laryngectomy in such a case when there is no definite microscopic evidence of malignancy. Even if some metamorphosis that indicates beginning malignant changes is taking place, we will hesitate to do a laryngectomy, since the lesion is superficial, not infiltrating, and both cords move normally. These cases sometimes do well under radiation therapy (see Cases 10, 12). Some recommend a partial tumor dose; others give a full tumor dose, according to the pathological picture and the progress during the therapy. Some laryngologists state that the vocal cords return to their normal appearance, but that has not been my experience. The cords seem to be thickened and white, and one must keep these patients under close observation for recurrence of a hyperkeratotic lesion. It is felt that partial doses are less likely to cause mucosal changes within the larynx, and the cords approach the more normal appearance. One wonders whether we should chance the partial dose. Under radiation therapy one can never be sure of a cure, certainly not so sure as when a lesion is removed surgically. We repeat then, a case of hyperkeratosis that would require a laryngectomy for complete removal is first treated by X-ray.

Occasionally one encounters a case where an entire cord is involved, and the growth extends across the anterior commissure for a short distance on the other cord (see Case 7). This can be treated by doing a bilateral thyrotomy, as suggested by Kemler, or the partial anterior laryngectomy as suggested by Jackson.

The cases that are puzzling are those known as pachydermia of the larynx, where the interarytenoid area and part of both vocal cords are involved. To eradicate this disease surgically, one would require laryngectomy. These patients are best treated by stripping the involved areas, by eliminating the irritating causes, and by close observation. If any malignant changes do occur a laryngectomy becomes necessary.

There is an occasional case where the tumor becomes very extensive, and, even though on repeated biopsy they are reported hyperkeratotic lesions, they should be treated by

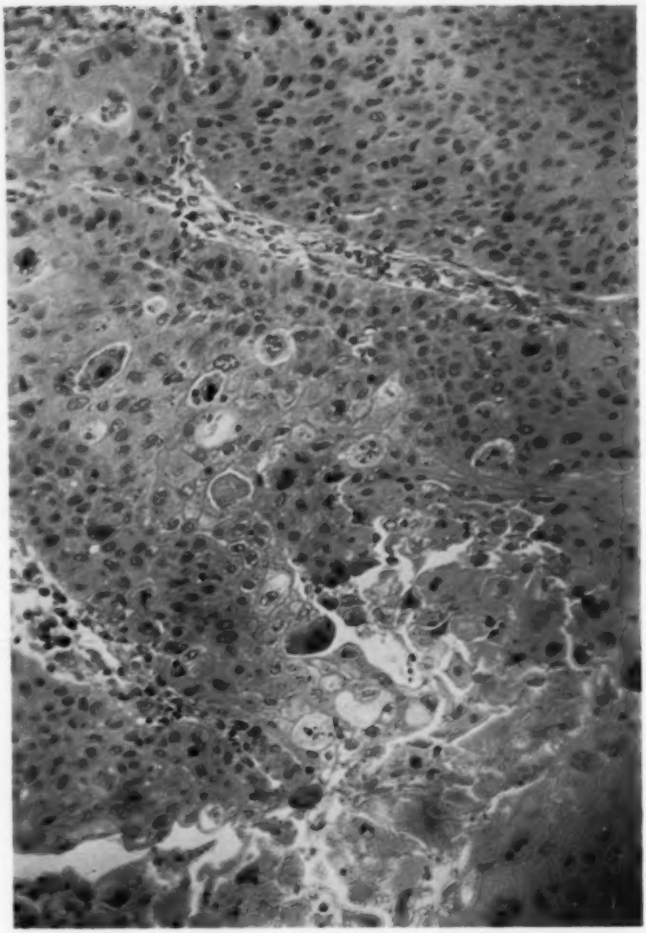

Fig. 4. Squamous cell carcinoma, showing the characteristics of carcinoma mentioned in Fig. 3.

laryngectomy (see Cases 9, 11). Recently we had the experience of procrastinating on just such a case because we found no evidence of malignancy microscopically, but clinically it was a carcinoma: it spread down the trachea, involving the tracheal stoma, infiltrated the tissue in the neck, and resulted fatally.

CASE REPORTS.

Case 1. M.A.—White, male, age 70. Hoarseness for many years. There was a red thickening on the anterior two-thirds of the left vocal cord. This area was stripped under direct laryngoscopy. The microscopic diagnosis was keratosis with some atypical cells; no evidence of malignancy. This patient has been under observation for three years, with no recurrence.

Case 2. E. M.—White, male, age 42. History of hoarseness for three months. There was a small irregular tumor in the anterior third of the left vocal cord. This was removed under direct laryngoscopy. The diagnosis was hyperkeratosis of the vocal cord with no evidence of malignancy. The patient was advised to undergo a regimen to eliminate irritation of the larynx. He has remained free from recurrence for over a year.

Case 3. S. H.—Age 59, W. F. The patient had a history of recurring attacks of laryngitis. The last attack had been present for four months. On examination she had a red, dry, granular appearing vocal cord. The diagnosis was laryngitis sicca. The patient had a chronic sinusitis, postnasal pus and atrophic appearing mucosa of the pharynx. On direct laryngoscopy there was thickened, irregular mucosa involving the arytenoid area and the vocal cords. The microscopic diagnosis of the tissue obtained was hyperkeratosis; no evidence of malignancy. The patient improved on treatment of the chronic sinusitis. It is important to point out that in this case there were no atypical cells and, therefore, it would not be considered a precancerous lesion.

Case 4. J. T.—Age 60, W. M. In May, 1955, he gave a history of hoarseness for three weeks. He had a lesion involving the left vocal cord; it appeared white and irregular. On direct laryngoscopy the cord was stripped. The microscopic diagnosis was keratosis with cell atypism of the left vocal cord. No evidence of malignancy. In May, 1956, the patient again complained of hoarseness for two months. There was a whitish superficial lesion on the right vocal cord extending from the anterior commissure to the middle third of the cord. This area was stripped under direct laryngoscopy. The microscopic diagnosis was hyperkeratosis of the right vocal cord with slight atypism but no changes indicative of carcinoma at present. In June, 1956, there was a recurrence of the lesion that involved the middle third of the right vocal cord; the diagnosis was hyperkeratosis with granulation formation. This patient has been under observation and there has been no evidence of recurrence to date.

Case 5. A. S.—Age 69, W. M. This patient gave a history of hoarseness for three months. He had a large keratotic-appearing lesion on the right vocal cord and subglottic area. Several specimens for biopsy revealed hyperkeratosis with suspicious areas of cellular activity and considerable inflammation. Because this was a warty, irregular appearing lesion that involved the subglottic area a laryngofissure was performed with removal of the entire growth. The pathological diagnosis of the laryngofissure specimen was carcinoma *in-situ*, in a hyperkeratotic lesion.

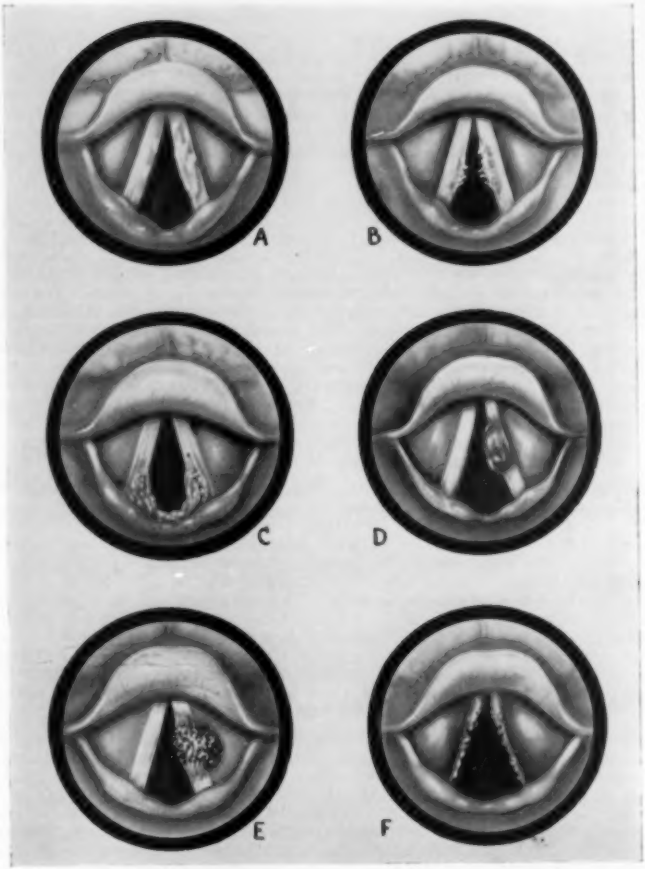

Fig. 5. A.—Leukoplakia; B.—Keratosis; C.—Pachydermia; D.—Hyperkeratosis with carcinoma **in situ**; E.—Hyperkeratosis with carcinoma **in situ**; F.—Bilateral Hyperkeratosis.

This patient, unfortunately, died of carcinoma of the right upper lobe with metastases a year later.

Case 6. M. C.—Age 57, W. M. This patient had five endolaryngeal removals of polypi and keratotic lesions. Two years prior to admission, he had had a tracheotomy at another hospital. At this admission he was having respiratory difficulty. He had a large grayish white cauliflower lesion in the glottic lumen, apparently arising from the anterior third

of the left vocal cord. After the preliminary tracheotomy, a laryngofissure was performed and a sharply circumscribed, elevated whitish lesion was removed from the inferior surface of the left vocal cord and subglottic area. The pathological diagnosis was hyperkeratosis of the larynx with no anaplastic changes noted. The patient has remained well to date, four years. This is an example of persistent recurrence of a laryngeal hyperkeratosis, and the growth of the lesion to such an extent that it gives laryngeal obstruction. It is significant, too, that no evidence of carcinoma was found, even after the laryngofissure.

Case 7. P. P.—Age 60, W. M. In April, 1955, complained of hoarseness for several months. He was a heavy smoker and drinker. On examination a whitish lesion was found covering the anterior two-thirds of the left vocal cord, reaching the anterior commissure. After a direct laryngoscopy, the biopsy revealed a keratotic lesion of the left vocal cord with some cell atypism; malignant transformation not evident. Because of the appearance of this lesion, a laryngofissure was done. The left vocal cord was removed, and since the lesion extended across the anterior commissure on to the right vocal cord a small portion of the latter was removed. The diagnosis was left vocal cord leukoplakia with carcinoma *in situ*; the right vocal cord showed leukoplakia of slight to moderate extent. The patient has remained well, though he has a small web in the anterior commissure. This case illustrates the fact that if the lesion is large and irregular, and when the microscopic section shows some atypism, it is best to do a laryngofissure immediately. A carcinoma *in situ* was found on complete removal of the growth.

Case 8. H. S.—Age 49, W. M. This patient complained of hoarseness for six months. There was a lesion on the middle third of the left vocal cord. It was granulomatous, grayish-white in appearance. It was removed by direct laryngoscopy. The microscopic diagnosis was hyperkeratosis of the vocal cord. There was evidence of inflammation in the underlying tissue. Three weeks later there was a recurrence of the lesion in the left mid-subglottic area. This was removed, and on microscopic examination revealed a carcinoma *in situ*. Two weeks later a laryngofissure was performed and the diagnosis was epidermoid carcinoma, very early, showing slight infiltration of the underlying tissue. This is a case of hyperkeratosis in which the metamorphosis into a carcinoma occurred rapidly or perhaps, as is more likely, the tissue was taken from the areas where the carcinoma did not appear.

Case 9. M. D.—Age 55, W. M. This patient appeared June, 1956, with a complaint of pain on the right side of his neck. There was an enlarged node over the carotid on the right side. On laryngeal examination there was a superficial grayish ulceration on the posterior surface of the right side of the epiglottis and right aryepiglottic fold, extending down to the anterior commissure. The left vocal cord was a little roughened; biopsy was taken. Diagnosis was hyperkeratosis with a few atypical cells; no evidence of malignancy. Since a carcinoma was not definitely found in the larynx, we decided to remove the enlarged lympho node. This was reported as chronic lymphadenitis. In Sept., 1956, the lesion looked about the same except that it seemed to extend on to the right vocal cord. Another biopsy at this time revealed carcinoma *in situ*, developing in hyperkeratosis of the larynx. A laryngectomy was performed, and the diagnosis was squamous cell carcinoma of the larynx; microscopic examination of the vocal cord itself showed hyperkeratosis. This patient has remained well for over a year. The case illustrates again that the biopsy may be taken from an area that does not reveal carcinoma. When one is suspicious of carcinoma the biopsy should be repeated.

Case 10. E. G.—Age 49, W. F. This patient complained of hoarseness for two years. Both vocal cords were thickened and dry; the right ap-

peared more involved than the left. The interarytenoid area was also thickened and irregular. The tissue removed from the larynx revealed a hyperkeratosis of the vocal cord and interarytenoid area with slight atypism. This patient was referred for radiation therapy. She has remained well for two years.

Case 11. J. G.—Age 75, W. M. In December, 1954, this patient had ulcerated appearing vocal cords but no fixation of the cords. The cords were thickened, white and had an irregular, warty, appearance. The diagnosis on biopsy was leukoplakia with considerable cell atypism; not yet definite carcinoma. Two months later the lesion looked worse, and the biopsy revealed squamous cell carcinoma. A laryngectomy was performed, and the patient has remained well to date. It is possible that a case of this sort should have been treated by radiation therapy, but it was felt that this warty extensive lesion should be removed surgically.

Case 12. H. S.—Age 63, W. M. Jan., 1956, this patient had had hoarseness on and off for 15 years. The recent attack of hoarseness had lasted four months. He had a reddish thickening on the left vocal cord and a white plaque on the right vocal cord. A biopsy of the left vocal cord was reported as malignancy of a low grade. The right vocal cord was reported as leukoplakia. He was sent for radiation therapy and has remained well; however, he does have thickening of both vocal cords.

CONCLUSIONS.

1. Those hyperkeratoses that show cell atypism are serious because they are precancerous. Since it is sometimes difficult to tell when the lesion will become cancerous it should be treated as a potential malignancy.

2. The early lesions are treated by stripping the cords under direct laryngoscopy and eliminating irritation due to tobacco, alcohol, abuse of the voice, infection, etc.

3. Recurrent lesions on one vocal cord or large warty lesions on one cord that involve the ventricle and/or subglottic area are treated by laryngofissure.

4. Recurrent lesions on both vocal cords are treated by radiation therapy, and if unsuccessful by laryngectomy.

5. Extensive laryngeal involvement should be treated by laryngectomy.

I am indebted to Dr. George K. Higgins for his help in the pathological aspect of this paper.

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SYMPOSIUM.

RECONSTRUCTIVE NASAL SURGERY.

MODERATOR:

RONALD TAYLOR, M.D., Vancouver, B. C.

PANEL:

J. H. OGURA, M.D., St. Louis, Mo.
HAROLD OWENS, M.D., Los Angeles, Calif.
R. I. WILLIAMS, M.D., Cheyenne, Wyo.

DISCUSSION:

I. B. GOLDMAN, M.D., New York, N. Y.
A. W. PROETZ, M.D., St. Louis, Mo.

PROBLEMS THAT CONFRONT THE RHINOLOGIST.*†

JOSEPH H. OGURA, M.D.,

St. Louis, Mo.

INTRODUCTION.

To many otolaryngologists, the subject of reconstructive nasal surgery sounds broad and includes all phases of rhinoplasty. Actually rhinologists who perform this operation use this term in a more limited sense. Total nasal reconstruction, such as the use of forehead flaps for the replacement of partial or total loss of the external use, is in the realm of the plastic surgeon. One may justly wonder then, how this fits into our field of rhinology? The otolaryngologist, by training and everyday practice, has always concerned himself with the

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function and health of the nose. A few, by greater experience, have become more involved with plastic surgery in its strictest sense. Thus, a casual observer may consider a rhinoplasty as a purely cosmetic procedure, and naturally wonders about the functional improvement claimed by such an operation.

I shall try to cover briefly four questions in a general way:

1. What do you do?
2. Why do you do it?
3. Do you accomplish what you claim physiologically?
4. How important is this?

It is not my intent to compare different schools of thought or to answer these four questions specifically, but to give a fundamental philosophical concept of handling a nasal problem.

It would be an easy matter to discuss this subject if the functional considerations for this surgery were clearcut. We owe a great deal of the present knowledge on the physiology of the nose, the nasal mucosa, turbinates and sinuses to the pioneer work of Jonathan Wright, Zwaademaker, Gray, Yates, Hartz, Proetz, Hilding and many others. Dr. Proetz has said that the normal nose is one that is symptom free, smoothly breathing with slit-like passages.[1] It is to him that we owe the understanding of how air currents pass through the nose.

On the other hand, such functional alterations as deviation of air currents and direction of flow ascribed to minor anatomical variations of the external nasal pyramid and lobule from the "normal" are not so well known. Some ideas have been expressed that traumatic injury which causes flattening of the lobule, and broadening of the base will convert elongated to round nostrils and may then alter air currents and cause symptoms.

It has been further expressed that the surgical correction of the lobule, or a deviated pyramid, back to "normal" offers

a more normal and harmonious relationship of the various anatomical parts with the objective of breathing better. Good anatomical restoration can be quickly determined by comparing a preoperative photograph with the operative result, but one cannot always demonstrate a functional improvement. The term function has been used all too loosely, and it may be invoked as a necessary prerequisite for a nasal operation.

No one will deny that when the septum obstructs one nostril, or is deviated so as to cause persistent impairment of the airway, surgical correction will give a satisfactory improvement in that patient. It does not necessarily follow, however, as far as very external nasal structure is concerned, that important physiological significance need be attached for every architectural aberration from "normal." An expression of an opinion is one thing, but facts documented on objective findings are another.

Our problem then, is just what is the normal nose? Are anatomical variations from "normal" abnormal for that patient? The restoration of an obstructed nose to "normal," to some may be more in terms of function; to others an emphasis on cosmetic appearance. The latter may be art, but it certainly is not science. The rhinologist is concerned with *breathing* or the symptomatic counterpart *nasal obstruction*. The restoration of such an airway is his interpretation of what an *adequate* airway should be. Since the nose cannot be compared to the highly developed sensory organ, such as the ear or eye, in that we cannot objectively measure what is taking place with respiration, we have no unit of measurement similar to the audiometer and speech test in audiology. One interprets the patient's complaint of nasal obstruction on the anatomical findings and can attempt to evaluate it only physiologically. This is our one criterion for surgery.

Since the impressive work of Proetz,[1] we have not advanced significantly in the field of physiology of the nose to the point where one can relate function with anatomic changes of the entire nose. We have made remarkable advances in techniques of rhinologic surgery in terms of preservation of turbinates, and nasal mucosa and avoiding radical sinus

surgery, but as yet we have no way of measuring nasal function in relation to the lower respiratory tract. There are rough methods for measuring airway by breathing upon a cool, polished metal surface, or more refined methods using the nasometer or rhinometer. We have taken measurements in various rhinologic diseases where nasal obstruction is present by using the rhinomanometer.[2,3] While the latter observations are important, this does not tell us too much about the physiology of breathing or respiration from the nose to the lung. Simultaneous observations on the nose and lung from a functional point of view that are being made under controlled laboratory situation, show that there may be some important relationship.[4] Why is it that a person with chronic lung disease complains of nasal obstruction, and his symptom of dyspnea is relieved if he can breathe better through the nose? Why does the occasional patient with a badly obstructed nose complain of dyspnea? Such problems are now under investigation.

The lack of objectivity in evaluating results (other than cosmetic improvement), the marked variation in complaints in patients with the same anatomic deformities (the psychosomatic and psychiatric aspects), the poor standardization of language among rhinologist and plastic surgeons, and the lack of a unit of measurement which is meaningful in terms of function, make it difficult to communicate with members of the profession, let alone our own colleagues in the field of rhinology. This does not mean that we are not trying to explore means of solving these perplexing questions. Measurement of architectural changes with growth and by trauma using nasal indices, cephalometric laminography are being investigated at the present time. One could assume the view of simplifying function in terms of our knowledge today, that the nose is an airway, air conditioner, filter, humidifier and an olfactory organ.

Our problems are quite similar to the field of orthodontia, and we are in a position of trying to establish this phase of rhinology as a science. Orthodontia has developed greatly, and after years of precise observations, evidence has accumulated that alterations to the teeth in the child in no way pre-

dictably changes the growth of the maxilla or the mandible. The restoration of teeth to their proper anatomical relationship cannot, to this day, be related to their function.[5]

With such limitations from the accepted functional point of view, it is obvious that the criteria for restoration of the nose to their proper anatomic relationship will have to be explained on other factors. The feeling seems to be prevalent that in a patient with a deviated septum or in another instance with a hump, physiologic needs are involved in order to justify the performance of a rhinoplasty. While in some instances this is necessary, in many other instances this will not be true.

The history of septal surgery reveals that the submucous resection, as advocated by Freer and Killian, was a satisfactory procedure when performed in certain types of deviated and obstructed septum. It was the functional and cosmetic complications, perforations, immediate and late saddling, and the retracted columella that was feared. Some of us knowingly, up to the 1940's, avoided operating upon a badly twisted septum, particularly when associated with a severely deformed external nasal pyramid with a tendency to sag on the dorsum, for fear of making this worse. If we had more courage we still did an incomplete septum operation, and we had difficulty in obtaining a satisfactory airway. If the patient asked for a repair of the hump, our deficiencies in handling this problem meant two separate operations, or calling in a plastic surgeon to handle the external nasal problem at the same time.

In the deviated nose, it is recognized that one cannot adequately straighten the septum without operating on the external nasal pyramid. Versatility of surgical approach for handling such problems is dependent upon the presenting anatomic problem.

When the patient complains of unilateral persistent nasal obstruction and the septum obstructs this airway, this can be easily understood; on the other hand, there are just as many patients who have a badly traumatized nose with septal obstruction without any complaints. Some atrophy of the interior of the nose may be present, and in this instance surgical correction may be indicated, even without complaints. One

explains the absence of complaints on the basis of the marked "compensatory process" of the nose. Combined septal and external pyramid surgery in another patient with the identical deformity, but with the chief complaint of nasal obstruction may be warranted. This patient may have subconsciously wanted his hump removed. When one obtains a satisfactory esthetic and cosmetic result, the patient at the conclusion of the post-operative period may only comment, "I can now breathe better." To this day, this is about the only criterion we have for improving this patient from the physiologic point of view. Of course, the patient will also be satisfied with his appearance, but one well knows how unreliable it is to depend upon what the patient says about his improvement of breathing and disappearance of many other secondary complaints. Infrequently nasal obstruction is still present, in spite of what appears to be an adequate airway and satisfactory cosmetic operation.

In some instances we can relate nasal obstruction to deformed upper lateral cartilages ("ballooned or collapse of the upper lateral cartilages"), and surgical techniques have been devised for preserving and correcting the so-called "valve" area. Alae collapse in moderate forced inspiration has been seen, and pressure measurements in such instances have been made.[3] Here surgical correction will require a total nose operation. Total nasal procedures are necessary to correct septal perforation, utilizing the principle of preserving nasal mucosa and rotating tube flaps from under the upper lateral cartilages and from the floor of the nose.

The problem of nasal obstruction in many cases is similar to that of the deaf patient. Some deaf patients get along very well and do not want or need surgery. Likewise, many patients with obviously obstructed septums do not complain of nasal obstruction, nor do they desire surgery.

The indications for surgery on the total nose, where the septum is obstructive may be done for one of two reasons:

1. To restore a proper airway which may entail cosmetic

change. Some cosmetic changes may not be requested by the patient. Some patients desire this change subconsciously or only if brought to their attention. In others cosmetic changes may become evident during or after surgery.

2. For cosmetic purposes.

It is assumed that the surgeon has taken precautions for not operating upon patients with psychiatric problems. It is particularly hazardous to operate upon patients who use their noses as a "crutch."

Certain deformities of the nose, associated with the complaint of nasal obstruction, require surgery upon the septum as well as the external nasal pyramid. The prize fighter's nose is a good example. In another instance, a twisted external nasal pyramid will prevent a deformed septum from being brought into its proper relationship unless the pyramid is restored to the midline. There are other examples. The patient may desire only a cosmetic change, and surgery to the septum is necessary if one is to restore the nose to the midline. It is recognized that the septum is the main supporting structure of the nose, and procedures which interfere with the septum the least, by gridding, cross-hatching, moving the maxillary crest to the midline, maintaining attachment of mucoperichondrium to one side and suturing the cartilage to the periosteum, it is believed that subtotal removal of the cartilage yields the best results for the septum and cosmetic appearance. The more extensive the septum surgery the more difficult it becomes to achieve the desired cosmetic result. This is particularly true when the caudal end is removed. Immediate saddling may result, but this can be avoided or minimized by subtotal removal.

Evaluation of the result is difficult when one is confronted with another type of problem. A satisfactory cosmetic result is obtained with an obvious narrowing of the nasal airway. The patient is quite happy with the appearance of his nose and he has no complaints of his airway. This is sometimes

the case when the patient's only presenting complaint is "to have the hump removed."

From the cosmetic point of view it becomes quite apparent, as one performs more of these operations, that one cannot become critical of the cosmetic or functional result obtained by other surgeons. Beautiful or mediocre results depend upon the initial problem; furthermore, what looks like a satisfactory result may change with time. Following surgery minor trauma may create further architectural deformity to the nose, such as a slight sag of the dorsum and a retraction of the columella. What is most important in any given case is that the operator can predict prior to and during the operation what may happen, anticipate such complications and institute appropriate corrective measures.

The decision has to be made as to whether to divide the operation into two procedures or whether it may be preferable to combine the operation as a one-stage procedure. When one interprets in terms as to what is best for the patient, it is always better to do the operation in one stage if possible. I cannot say that the plastic surgeon is concerned with cosmesis alone, and is not interested in the airway in all instances.

The position in which the otolaryngologist finds himself is obvious. A properly trained rhinologist should be the logical one to cope with a problem of this sort, which is complicated by consideration of improvement in function, cosmetic appearance and social well-being of the patient.

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HOW DO YOU GO ABOUT MAKING A DIAGNOSIS IN SOME OF THE MORE IMPORTANT RHINOLOGIC DEFORMITIES?*†

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If any one thing comes to mind it would be to say that making a diagnosis encompasses the whole totality of the nose, both inside and out. One cannot merely say that the patient has a hump or a saddle, or the nose deviates. We must know all the parts of the nose which produce the deformity and their relation to each other.

To arrive at a diagnosis, some arbitrary classification is necessary in order that the surgeon can decide what to do. With very few exceptions, one should have the diagnosis pretty well made and the operation mentally done before entering the operating room; however, the diagnosis may not be complete until the patient is anesthetized, or until part of the surgery has uncovered the deformity.

A working classification for diagnosis should encompass the following:

We are interested in the texture of the skin, whether it be thick and oily, thin and atrophic, or of relatively normal appearance:

Thick, oily skin often interferes with proper narrowing of the nose, particularly in the lobular area. An extreme example of this is a patient I operated recently who had had both lateral crura removed at a previous surgery and yet the tip was still quite thick, and there was no collapse of the nares. Thin, atrophic skin offers problems of contractural deformities after surgery.

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Septal obstruction with minimal or no external nasal deformity:

Obviously this is a personal matter with the physician. A Killian or Freer septal procedure is quite adequate, and no external nasal pyramid surgery is needed.

Septal obstruction with a dislocation of the caudal end of the septum off the nasal spine and premaxilla into the vestibule with minimal external deformity:

This type of deformity will need a subtotal, or total removal of the septum, including a complete mobilization of the caudal end of the septum in the maxillary spine area. External pyramid surgery may or may not be indicated.

The deviated or twisted nose, usually of traumatic origin:

Such a nose has been discussed fully by Huffman and Lierle.* This type of nose runs the gamut of diagnostic problems. One should note the deformity of the dorsum, or more correctly the sagittal alignment. Is it "C" shaped, "S" shaped, or merely deviated. Sagittal alignment with other structures of the face and teeth must be considered. Other accompanying deformities of the maxilla and teeth might propose the question whether a nose should be replaced in the midline.

The nasal bones are usually uneven and asymmetrical, and there may be a hump. These conditions are of prime importance in determining hump removal and the level of lateral osteotomies.

The relationship of the upper lateral cartilages to the nasal bones, as well as the septum must be determined. Often the deviation may involve only the cartilaginous vault and septum, and correction of this area is all that is necessary.. The upper lateral cartilages may be torn from their attachment to the nasal bones, and present inequalities.

The lobule-septal relationship should be noted. Is the twisted tip and columella due to septal dislocation or it is due to lobular deformities, particularly the medial crura?

*Huffman, W. C., and Lierle, Dean M.: "The Deviated Nose." Ann. Otol., Rhinol. and Laryngol., Vol. 63, Mar., 1954.

The deviated nose offers the most difficult problems in diagnosis and management, as it requires the mobilization of the septum and external nasal pyramid as complimentary procedures, and in most cases the procedures should be done at the same time. This type of nose should be the great concern of the rhinologist.

A high narrow nose with an excessively long septum:

This type of nose often appears like the tip is hanging on the end of the septum and is most pronounced when the patient smiles or purses the lips. One should note the lobular cartilage development, as the lack of projection of the tip may be more apparent than real, and all that is necessary is to decrease the projection of the bony and cartilaginous dorsum; this must include the dorsum of the septum also. The nose actually must be widened.

We sometimes see a low flat nose with a deviated caudal end of the septum:

This type of nose usually results from poor tip development as a result of childhood injury, and most of the pathology will be in the cartilaginous pyramid and septum. One should decide whether the cartilaginous saddling is due to loss of septum, atrophy of the upper lateral cartilages, or both.

The caudal septum may lie almost at right angles in the nose, or be quite short. The latter is often accompanied by a poor maxillary spine and retraction of the naso-labial angle. These areas must be carefully studied, as the correction of this deformity usually requires the use of implants of various types.

The straight humped nose with or without a drooped tip offers little problem:

This is the so-called "cosmetic type" and can be and is handled by a typical Joseph rhinoplasty. The only problem is that the septum should be studied, as many of these patients have a good cosmetic result, but they will often tell you they cannot breathe well after the surgery.

In any work of this type the psychological diagnosis is important:

A large percentage of patients come to us merely because they want cosmetic improvement, although this may not be the presenting complaint. Many of these patients have used their nasal problem as a crutch, and we must be careful not to remove this crutch and in so doing possibly convert a psychoneurotic into a psychotic personality.

These are just a few of the problems to consider in arriving at a diagnosis of basic nasal deformities.

SURGICAL MANAGEMENT PHASE.*

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It is my deep conviction that it is in the best interest of all that one school of thought not be pitted against another. It is rather my belief that a discussion will bring to all of us a better understanding of the many facets of this perplexing problem. In turn, this will stimulate study and advancement in our field of rhinology.

From a broad, philosophical standpoint, our concept considers the total nose, the total relationship of all of the components of the nasal septum, the external nasal pyramid and the internal nose; therefore, the surgical procedure for restoration of proper anatomical relationships must, of necessity, not be limited to only one portion of the nose.

We all agree, I believe, that the nasal septum is the main supporting structure of the external nasal pyramid. Modifications of the nasal septum create problems in direct proportion to the extent of disturbance of septal totality. With extensive disturbance, complications such as saddling, retraction of the columella, and widening of the nose may occur.

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Many of us have observed these complications during and following surgery. It is our belief that these complications should be anticipated, and methods of correction instigated during surgery and, of course, avoided if possible.

The versatility of the approach by application of rhinoplastic techniques, not necessarily original, to septum surgery, the preservation of the mucosal flaps and preservation of the skeletal portions, as well as the soft structures, allows the freedom to perform surgery on the nasal septum and the adjacent external nasal pyramid and, in many instances, the internal nose, in one surgical procedure. This flexibility, in my experience, allows us to modify and attempt to restore proper relationship of the component structures, and to restore totality to the best of our ability. This results in a better nose from a cosmetic aspect, and allows the patient to breathe better.

Now, what is a normal nose? Dr. Proetz[1] defines the normal nose as follows: "The normal nose, or perhaps better the symptom-free, smoothly breathing nose, is characterized by slit-like passages—slit-like everywhere and not merely above, in the olfactory area."

The normal nose is difficult to define because each individual nose has its own peculiar characteristics, structural variations, as well as functional, when we consider the total personality.

We are all aware of the tremendous inherent compensatory powers of the nose. We are also aware of variations which in one person may produce symptoms, in another they may not, and on clinical examination very little difference can be observed in the structures of the nose.

We do feel, however, that there should be a proper relationship of the vestibule to the *os internum* of the nose, and also a proper relationship of the lateral walls of the nose to the nasal septum. Here again it is difficult to put into figures just what this relationship is.

Studies of the various indices, such as the choanal index and the clinical nasal index, as well as the X-ray nasal index,

give us figures which are only relative, but which do help in evaluation to some extent.

The attitude of the nostrils plays an important role. When the nostrils are round and wide, usually the *os internum* is similar, and an excessive amount of space is usually present between the lateral nasal wall and the nasal septum. We are particularly concerned with injuries to the Caucasian nose which may simulate, in many respects, a nose of a different ethnic origin. A study of embryology is quite revealing in this respect; neither can anthropological considerations be ignored. In reconstructive surgery, an attempt is made to restore these relationships to what we feel is normal for that particular nose.

What do we mean by "breathing" and what is an "adequate airway?" My conception of an adequate airway is one in which there is not an excessive amount of space in the nose from the nares to the posterior choana, but rather one in which the passages are comparatively narrow and streamlined. One is struck by the fact that the passages are slit-like, and many structural prominences are noted which seem to encroach upon the lumen. Breathing is an esthetic sense, and to date we have not found a satisfactory definition.

Our knowledge of the exact function of the entire nose or, for that matter, the exact function of any integral portion of the nose is limited. We feel that the nose should function as a unit and complement the other structures of the respiratory apparatus. To my knowledge, we do not have a satisfactory explanation clearly establishing the nose-lung or the mouth-lung relationship.

It is true, that anatomical shapes and relationships may be mistaken for functional necessity, but again as Dr. Proetz[2] mentions, the structural variations may be useful as a starting point, providing that we recognize our limitations.

Deformities of the external nose are frequently the result of injuries or developmental abnormalities. The deformities may involve any or all parts of the external nasal pyramid and the nasal septum.

To reconstruct and to restore, or provide totality to the best of our ability, it may be necessary to separate the component structures, either partially or completely, and to move and reposition, or remove and replace the component parts.

The modifications of the septum may require conservative or extensive procedures. In either case, it should be feasible to accomplish the result with, *1.* minimal injury to the mucosa; *2.* with rigidity restored to the nasal septum; *3.* with sagging of the cartilaginous dorsum prevented, or corrected if it was present or occurred, and *4.* with prevention or correction of post-operative or post-traumatic widening of the nose.[3]

The exposure to the septum should afford us opportunity to observe adequately and to perform surgery on any portion or portions of the nasal septum.

Frequently the septum operation alone is inadequate to restore proper anatomical relationships. We will concern ourselves with four categories of deformity which may require attention in conjunction with the septum operation:

The first category includes those which require surgery of the lobule in addition to modification of the nasal septum.

The second category involves those which require surgery of the external nasal pyramid, but in which a hump removal is not needed. A good example of this is the injured nose that is leaning to one side and possibly twisted.

The third category comprises those patients who require rather extensive modification of the septum, removal of the hump, modification of the cartilaginous vault and the lobule. The classic example is the severely twisted or deviated nose.

In the fourth category, the typical boxer's nose would be the classic example. This nose would require extensive septum surgery, moving of the bony external nasal pyramid, modifications of the cartilaginous vault and lobule.

Preservation of the mucosal flaps and their attachments in entirety or in part, makes the combined operation possible.

In addition to this, if possible, preservation of the attach-

ments of the upper lateral cartilages and the attachments of the nasal bones to the septum, help support the nasal pyramid. If at all possible, we like to leave a portion of these structures attached to the septum; however, if we have an intact mucosal tunnel, by preservation of the mucosa, the intranasal dressing will serve to hold the structures in position for adequate healing.

These attachments may be severed and still preserve the mucosal flap by separating them intraseptally or extranasally. The nasal bones may be separated from the bony septum in a similar manner. If the septal flaps have not been elevated, it is possible to perform a rather extensive submucosal elevation of the upper lateral cartilages and the undersurfaces of the nasal bones. The upper lateral cartilages and nasal bones can also be separated via this approach. This procedure is also rewarding in attempting to close a septal perforation by merely continuing the elevation from the undersurface of the upper lateral cartilages and the nasal bones onto the septum, and allowing the mucosa to drop, and also raise the mucosa from the floor of the nose so as to close the hole of the perforation.

There are many procedures which can be used to remove the hump. Certainly the saw is very effective; the chisel is likewise effective. Other methods have been advocated and they are very useful.

We believe, however, that by not being bound by any procedure or routine, the flexibility of approach, the preservation of mucosal flaps and the selective use of various techniques, in part or in whole, at will, and as may be indicated in a given circumstance, enables us to gain our ultimate objective.

In summary, our concept is in no way original. It involves the application of rhinoplastic techniques, providing us with a versatile approach to many variable problems encountered in reconstructive surgery of the nose.

Preservation of the mucosal flaps and preservation and modification of the structures provide us with a method whereby restoration of anatomical relationships can be ac-

complished and the totality of the nose restored to as near normal as possible.

We do not know all the answers to the surgical phase. Neither do we know the answers to the functional phase. We are critically reviewing our results and hope that this will lead us to the solution of the many problems with which we are faced.

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DISCUSSION.

Irving B. Goldman, M.D. (New York, N. Y.): I am in accord with the basic philosophy of all the panelists and agree that total reconstruction of the nose be left to the general plastic surgeon, where restoration is primary and function secondary. The problem of nasal physiology I shall leave to Dr. Proetz. I should prefer to consider the practical aspects of rhinoplasty.

Before I do so, I should like to discuss the antagonistic attitude to the term "rhinoplasty" by many physicians and surgeons. The various synonyms require reevaluation of what constitutes the correct term for "cosmetic" plastic surgery of the nose.

Some of the names and phrases indicating the same procedure are: rhinoplasty, nasoplasty, nasoseptopexy, reconstruction of the nose and reconstruction of the nasal pyramid. Some surgeons classify the same procedure under three different headings. This is confusing to the operating room staff as well as to the assisting resident surgeons; as a matter of fact, in some hospitals the use of the word "rhinoplasty" is forbidden—taboo. In filling out charts, the surgeon must state he performed a nasal reconstruction when actually he had carried out a corrective rhinoplasty.

Why should we, at the present time, conceal correction of external nasal deformities which are so frequently associated with internal ones, under such a great variety of nomenclature? The term "rhinoplasty" should be used today without verbal camouflage. The patient understands this terminology much more than the phrase "reconstruction of the nasal pyramid." Many a patient spontaneously declares that "I have had a rhinoplasty."

The term "rhinoplasty" is derived from the Greek "rhis"—nose, and "plassein"—to form. It is obvious then that a change in the form of the nose or any component part, constitutes a rhinoplasty. It does not matter whether it is restorative, corrective, cosmetic, complete or incomplete. In other words, it is the correct term for the operation, despite its questionable implications.

It has been my policy when teaching corrective surgery of the external nose to use the term "rhinoplasty." I was greatly surprised to learn that physicians from different parts of the country objected to the use of this word. The objection was not personal, but rather to the effect this would have on their medical colleagues. It is obvious that in the various parts of the country they must conform to the usage of the term "rhinoplasty." It is a simple inclusive one. Its ancient implications have been disspelled. It has taken on a dignified and well merited status. The art has reached a high degree of development. The scientific journals welcome articles on rhinoplasty, and medical societies accept papers on rhinoplasty, indicating that the term is becoming a widely acceptable one.

The basis of all corrective rhinoplastic procedures is preoperative diagnosis. Analysis, relying on the rigid use of measurements, laws, and formulas, cannot supplant taste, judgment, and experience, essential in fine nasal plastic surgery. As there are no two noses alike, no set of rules can be drafted that would fit all shapes of noses. Certain principles when applied to every nose, will disclose its abnormalities. The routine analysis considers forms, proportion, and harmony in relation of the nose to the rest of the facial features. The fundamental artistic cannons must be fully understood by the rhinoplastic surgeon who is seeking a good esthetic as well as functional result.

In planning to correct any nasal deformity, careful attention must also be given to the nasal septum which has a great bearing on the method of operation. Is the septum a minor or major factor in the rhinoplasty? What should be the method of approach? The intranasal examination is supplemented by palpation of the septum. This may reveal thickness and distortions.

Planning begins with visual examination. Is the correction feasible? Is the operation corrective or does it require much reconstruction, *e.g.* a harelip nose, bifid nose, a dish-face deformity? Has the operation limitations, *e.g.*, a short columella or an atrophic lobule, or very thick skin? The surgeon must plan to compensate; where to over- or undercorrect.

The visual examination is followed by palpation to determine the size, position, and character of the nasal bones, the form and position of the upper lateral cartilages, the texture of the skin, and shape of the lower lateral cartilages.

The nasofrontal angle may not have a glabellar depression. Palpation may reveal a thick procerus muscle. A roentgenogram of this region is advisable to determine the depth of the bone and the relation to the frontal sinuses.

Palpation at the base of the nose in case of a naso-labial web may reveal a projecting nasal spine or the curved overhanging margin of the septal cartilage. A recessed naso-labial angle may be more retracted when pressure is made with the examiner's finger or instrument. A retrusion caused by absence of septum contraindicates a complete transfixion; on the other hand, restoration of the naso-labial angle in the congenital variety requires a complete separation of the membranous septum from its septal attachment for mobilization necessary for reconstruction.

Properly scaled photographs with a study of the nasal components in relation to the face, forehead, and chin are important for analysis, diagnosis, medicolegal and clinical records. Each view, lateral, frontal, nasal, may reveal a feature requiring correction. Nasal deviations and irregularities are better shown in color photography.

Photography is also important because it serves as a common meeting ground between the patient and the doctor. By reversing the photo and placing it in an illuminating box, the surgeon can easily draw the profile line which he considers esthetic. This may or may not meet with the patient's approval and is a good way to avoid pitfalls of operating upon a neurotic individual who seeks a nose not compatible or attainable with his or her features.

The diagnosis of a nose requiring revision of a rhinoplasty, although subjected to analysis, must be considered to be an exploratory operation. Not until all of the parts are surgically exposed, manipulated and reassembled, can the surgeon make his precise decision for correction, *e.g.*, in many patients I found that complete osteotomies had not been performed.

The wide nose creates problems because of the tendency to recurrence. If it is present as a racial feature, as in the Negro, an implant or graft, as a rule is required without performing an osteotomy. Otherwise, if the lateral walls are of insufficient height to cover the vault, one must decide whether an osteotomy and repositioning of the nasal processes of the maxillae has to be carried out prior to dorsal implantation.

Width, caused by excessive thickness of the nasal bones requires radical excision of these structures, mesially and laterally. I prefer to depress the nasal processes on mesial shifting after osteotomy in order that the septum, perpendicular plate and nasal spine of the frontal project in the midline and become the summit of the nasal pyramid, producing a narrow nose. Ventral surfaces of a thick septum are thinned and redundant tissues, such as mucous membranes lying between the bones and upper lateral cartilages are removed.

Correction of a nasal deformity associated with a deviated septum, frequently presents surgical problems. These range from timing of the septal operation to the variety of the operation to be performed. The questions which usually arise are: Should septal correction be carried out prior, during, or after the rhinoplasty? If a deviated septum is present, which operation should be performed to alleviate nasal obstruction and permit correct alignment of the nasal structures? Will a contemplated nasal profile and columella be placed in jeopardy with the production of a saddle of the upper cartilaginous vault, a dropping of the lower cartilaginous vault or collapse of the entire nose?

Complete resection of the septum and replacing it in part, based on a "tie-in" of the upper lateral cartilages with the septum, has not proved reliable. It was believed that the former structures alone could support the cartilaginous nasal dorsum, because they were an integral part of the septum and were attached subjacently to the nasal bones and the frontal processes of the maxillae. They were thus able to furnish adequate stability to maintain profile projection under static conditions. Nevertheless, removal of the cartilage and insertion of strips or resected cartilage did not prevent secondary cicatricial contraction and did not prevent an external dorsal depression or columellar retraction. The cartilaginous implants between the mucoperichondrial layers frequently became twisted, often to a degree that produced nasal obstruction.

Subtotal or complete resection of septal cartilage with insertion of implants between the mucoperichondrial layers may be required in a depressed variety of septal deformity. The sunken or fragmented septum cannot be shifted into its normal position and must be sacrificed. As a rule, however, this kind of septal deflection is already accompanied by an external saddling deformity. This would require insertion of dorsal grafts under any circumstances.

The procedure I prefer restores the normal anatomic relationship of the deflected septum without or with minimal sacrifice of its structures. The principles of the operation consist in mobilization of the septum to the midline by use of pedicle flaps of caudal septal sections, central replacement of posterior osseo-cartilaginous section, resection, if necessary, of a small cartilaginous or bony bulge which can be obstructive. No columellar retraction develops with this technique. Straightening the caudal distortion, and thus elongating the septum, frequently corrects the columellar retraction. Neither drooping of the nasal tip nor saddling occurs. The septum gives the nose the same relative support after it heals as one that has not been operated upon. It is, therefore, not so vulnerable to trauma in which septal components have been widely resected.

Both sides of the caudal part as well as the remainder of the septum must be freed. A unilateral or hemitransfixion does not liberate the distorted mucoperichondrial layers and thus prevents correct alignment of the replaced septal parts. Postoperatively, healing is interfered with, owing to traction of the unelevated mucoperichondrium. In consequence, there often is recurrence of the deformity. This becomes obvious if one considers the pathogenesis of the caudal deformities. Fracture of the cartilage occurs, and in the process of repair a fibrous invasion takes place between both sides of the mucoperichondrium and the cartilaginous fragments, and/or overriding fragments become intermingled with the mucoperichondrium.

Experience has shown a rhinoplasty following a previously adequately corrected septum may also be followed by collapse of the nasal dorsum. This occurs after osteotomy and meatal shifting of the nasal processes. The remaining osseo-cartilaginous structures, despite supportive intranasal packing, are unable to support the nasal dorsum. The earlier rhinoplastic surgeons, through sad experiences aware of this potential hazard, were wise to retain as much of the septum as possible in the carrying out of a concurrent rhinoplasty. Obviously, there were many examples where nasal obstruction had to be corrected subsequently.

The recent trend has been to perform a simultaneous rhinoplasty and submucous resection. The septal correction whereby the structures are mobilized but are essentially intact is, in my opinion, the answer to the combined operation. The upper lateral cartilages can be cut from the septum and appropriately trimmed.

A combined rhinoplasty and submucous resection is also preferred by the patient for economic reasons, for convenience, and for time saving factors and the avoidance of two hospital admissions. The rhinoplasty and an adequately performed septum operation can be concurrently accomplished with a good functional and assured esthetic result. This is of benefit to the patient and of advantage to the surgeon.

Certain nasal deformities require multiple procedures such as in children with saddle noses caused by trauma and/or infections, resulting in loss of septal support. Retrusion of the middle third of the face may form owing to lack of development. Preserved cartilage implants reinserted until full nasal growth helps build-up and raise the nose. Harmless, non-absorbably foreign material may be more useful. I inserted a large piece of ivory in a young patient whom I observed for ten years. The original ivory implant was so adequate that, at the age of adolescence, she refused further surgery and the nose was in good proportion to the rest of the face. Today I would consider using an acrylic or polyethylene implant in a child in order to avoid many operations.

Among the other conditions is the short columella. This is essentially a skin condition and elongating the inadequate columellar skin without

manifest scarring is difficult. After insertion of an implant on the columella, reinsertion and another procedure to lengthen it may be necessary.

The unusually small underdeveloped lobule, often accompanied by flaccid crura is difficult to model despite the insertion of implants. This is also a skin problem, as it cannot provide an adequate bed for the necessary implant. If the profile line cannot be lowered to meet the apex of the tip without producing a dorsal depression, increase in size of the lobule can be attained only by an implant or graft, after reconstruction of the soft parts.

Occasionally, the surgeon attempts a rhinoplasty in which he removes the hump without modelling the tip, or correcting the tip without modifying the osseous or upper cartilaginous vaults. A non-satisfactory result will require a revision with insertion of the omitted operative step. Flaring alae, not corrected at the time of the rhinoplasty, may require correction at a later date.

It is well known that among the other nasal conditions frequently requiring more than one operation, are the harelip nose, nasal atresia, dish-face deformity, and bifid nose.

Healing plays an esssential part in the final rhinoplastic result. It is common knowledge that a nose which appears perfect at the end of a rhinoplasty, or when the external dressing is removed, may assume an undesirable form as early as the tenth post-operative day. One may be asked: What is the nature of this process in tissue repair which militates against the welfare of the patient? Rhinoplastic surgeons have become aware of the risks of oil, thick and pre-acne rosacea varieties of the skin; nevertheless, sooner or later, the surgeon operates upon such patient and the skin "behaves" badly as healing takes place, producing an unsatisfactory esthetic result. Ideal situations for rhinoplasty are not always encountered.

The time has come for the otolaryngologist interested in rhinoplastic surgery, not to confine his thoughts solely to the technique or diagnosis. If he is to continue to advance rhinoplasty, he must undertake research in the process of skin healing, for on this hinges the more acceptable results.

The panelists have been using the word "crutch." To attain recognition of his position in rhinoplasty, the otolaryngologist has had to pave the way by exploiting various representations like nasal physiology, for example. The time is appropriate for the otolaryngologist to refrain from hiding behind subterfuge of disturbed nasal physiology as the single indication for his interest in rhinoplasty. It is generally conceded that the nasal septum requires correction in from 60 to 70 per cent of all rhinoplasties: rhinologic training, the skillful use of the head mirror or headlight, intranasal tools and correct manipulation are the *sine qua non* for an overall cosmetic rhinoplasty. The time has come for the rhinologist to declare that he is rightfully entitled to correct a nasal deformity for cosmetic and psychologic reasons. It should be made clear that a rhinoplasty is not performed solely for the preservation and restoration of nasal function. If a functional cause exists, this, of course, adds to the necessity for operative intervention.

Finally, it is my belief that too much emphasis has been placed on the ill-effects of intranasal incisions and preservation of the "valve" areas. Patients complain of nasal obstruction following a rhinoplasty when excessive vestibular skin has been sacrificed, causing synechase between the lateral crura and septum. Various degrees of alar collapse causing disturbance in breathing accompany the pinched tip. Although the septum and upper lateral cartilages caudally form the so-called "valve," severance

of these structures in a rhinoplasty is not followed by disturbed physiology after reattachment on healing whether the excessive cartilage is removed *in toto* or submucosally. Evidently, the scar tissue formed at the valve region does not appear to interfere with its presumptive function. It is only when the redundant protruding upper lateral cartilage is not removed that it acts mechanically to obstruct the airway producing nasal obstruction. With the removal of the excessive projection, adequate airway is restored. The important structure in rhinoplasty requiring preservation is the membranous septum. Sacrifice may cause columellar retraction and loss of service should a strut be required.

ARTHUR W. PROETZ, M.D., (St. Louis, Mo.): I have listened with great interest and attention to the presentations of my colleagues and have been at some pains to read as much of their writings as I could find, since my assignment was directed to the physiological aspects of the subject and not too much has been said on that score during the preceding discussions.

I have also brought along a specimen. Forty-five years ago this nose had a terrific spur removed from its septum, through a submucous resection, and parts of both inferior turbinates were trimmed away with scissors. Before and after the operation the ridge-pole ran in two directions and ended in an unsightly lump—and still does. Whether this deformity blighted its owner with an inferiority complex I must leave to you. Physiologically it is perfect.

There has been much controversy over the propriety of combined operations on the nose, and before launching on my theme, I should like to contribute one or two views from the sidelines:

1. If a nasal inlet needs widening or narrowing, or the septum straightening, and besides this the profile needs fixing—if, in short, combined operations are indicated, it seems axiomatic that they should be done at one time and by one operator. If done in two stages, the second operator is almost bound to be hampered by scar tissue and by someone else's leavings which are not part of his plan, and the patient suffers double pain and expense.

2. If Dr. John Smith is a competent surgeon, familiar with the specific anatomy, physiology, pathology and surgery of all the parts concerned, and has the necessary judgment, based on experience, then, and only then, he is the man for the job. All other qualifications combined cannot compensate for the lack of any one of these. After that, whether Dr. Smith is called a plastic surgeon, or a rhinologist, or a proctologist, could not matter less to the patient or his tissues. Jurisdictional disputes are as transparently commercial in this field as they are in others.

3. Let us honestly face the distinction between physiology and cosmetology and never, not ever, confuse the two for the purpose of making any surgeon, or any operation seem more "respectable" or more attractive, or for any other reason.

And so to work.

With the surgical procedures discussed here and their purpose, I have nothing but agreement and approval, in particular with some of Dr. Owens' straightforward remarks, within the limits of my experience. On physiological grounds I fear we part. In all frankness, I cannot get away from the feeling that there is a good deal of groping for some new scientific background to fit surgical procedures already current, which to me has all the disadvantages of walking backward, and has in the past led to too many untenable hypotheses.

We all agree that, as stated, a nose that isn't working needs fixing.

As to what to do and how far to go, Dr. Ogura laments the lack of any concise standardizing measurements such as we have for sight and hearing. I doubt that they would be of much help if we had them, for, as Dr. Ogura himself observes, the two cases are scarcely parellel. Only the olfactory portion of the nose is a special sense organ like the eye and the ear, and the sense of smell has not been brought up in this discussion. We are dealing with something much simpler and less critical. I have no doubt that anthropologists have scientifically measured and classified our hands and feet, (they come in all sizes, shapes and colors) but while anthropological indices are helpful in such projects as tracing our remote ancestors, we do not use them to find out how we all run, or dance, or write, or who can stretch an octave. It simply isn't that complicated.

We know that one asymmetry here is often compensated by another asymmetry there, and put together they make a healthy nose. That also is not too difficult to determine.

Although the shape of the nostril is less important than its size and direction, even in the last much latitude is permissible, and minor variations in the angle of tilt are of small consequence—physiologically, that is.

From the standpoint of warming and humidification, the vestibule is practically negligible, being too small and made of the wrong things.

The submucous resection of Killian and Freer has been mentioned as a "satisfactory procedure when performed in certain types of deviated and obstructed septums," but fears are expressed regarding cosmetic complications, perforations, saddling, and retracted columellae, as though these were common accidents. In incompetent hands they may be, but that is true of any operation. Perforations we have all had; most of them negligible, unless they whistled, in which case they were promptly repaired.

So long as I can remember, rhinologists worthy of the name have diversified the standard procedures to fit cases, and have variously revised faulty passages without ado. It is my observation that those who have had many unsatisfactory results from septal surgery have not been very good at it. While certainly not implying that complaints from the patient are the only justification for operating, I do not quite follow Dr. Ogura when he says (I copy him verbatim): "The feeling seems to be prevalent that a patient with a deviated septum or in another instance with a hump, physiologic needs are in order to justify the performance of a rhinoplasty. While in some instances this is necessary, in many other instances this will not be true." Unless Dr. Ogura is talking now of pure cosmetics I wonder what these many "other instances" could be.

I cannot agree that, as has been stated this morning, our knowledge of the exact function of the nose is unduly limited. Merely because a function is simple, we need not feel obliged to postulate something more complicated, and as yet unknown. I would say that our present knowledge serves us very well, and in the same breath enthusiastically encourages new discoveries and conceptions, but I would insist that they rest upon firm scientific foundations, and I should prefer that they were practical.

On last Friday I had a letter from Dr. Williams, saying that "for reasons of time limitation" he had decided to delete four pages of his presentation. By some unfortunate coincidence these were the four pages dealing with the physiological aspects which I am here to discuss.

As, however, they do constitute some of his thinking and that of his group, and I do have the time, I should like to quote some of the material he was obliged to skip, and to bring it up for discussion, since I think it too important to overlook.

If I misquote Dr. Williams in anything, I hope he will stop me.

Dr. Williams stated: "We are aware of variations which in one person may produce symptoms, in another they may not, and on clinical examination very little difference can be observed in the structures of the nose." Again, I wonder whether I interpret him correctly as proposing surgery on these symptomless cases when he continues: "We do feel, however, that there should be a proper relationship of the vestibule to the os internum of the nose, and also a proper relationship of the lateral walls of the nose to the nasal septum." I hope not, since he admits in his next sentence that "it is difficult to put into figures just what this relationship is" and that "the various indices, such as the choanal index, and the clinical nasal index, as well as the X-ray nasal index gives us figures which are only relative."

I cannot say whether I agree with some of the other statements or not, since I do not understand just what they are meant to imply. For eyample: "When the nostrils are round and wide, usually the os internum is similar and an excessive amount of space is usually present between the lateral nasal wall and the nasal septum." Excessive by what standards? Injurious? Symptom producing? Or merely larger than some ethnological mean? "We are particularly concerned with injuries to the Caucasian nose which may simulate, in many respects a nose of different ethnic origin." Does that mean that if my nose had the characteristics of a negroid nose it would not serve me as well as it would my African friend next door? Presumably it is meant that such a nose would be disproportionate to the rest of my Teutonic cranium and that its function would thereby be upset. This could be, sometimes, but the ethnical reference seems remote. Dr. Williams adds "A study of embryology is quite revealing in this respect," but he does not tell us what it reveals, or what is its surgical importance.

"Neither can anthropologic considerations be ignored," but he does ignore them in his discussion and it would have been interesting to know what they are and what they show.

Still on the physiology of the nostrils, I can summarize only by saying that I regard it as much more definite and much simpler than it is being painted. To put it in one sentence: "The function of the nostrils and the pyramid is to pass an adequate amount of air in a generally upward direction into the nose." I say "generally" upward because the angle is not very critical.

There have been recurring refrences to "philosophies, concepts, indices, ethnics," "the disturbance of septal totality" and so on. It all sounds very profound, and very scientific, and very new; frankly I cannot see it as being very much of any of these. When the fury of the tempest dies away it all ends up in a teapot and the teapot is a familiar piece of antique crockery described by Charles Bell a century ago, and by others since.

The teapot, also, is a fine example of the vestibule and how it works. If you will permit me, I will make a sketch of it (drawing on the board). The teapot is a hollow object spouting a jet. The nature of this jet is determined by the size and shape and direction of the opening at the tip and by nothing else. The teapot represents the vestibule and the opening represents what one has lately been calling the os internum, which, by the way, my dictionary defines as "the uterine end of an oviduct."

So long as the jet is directed *generally* upward things are all right. It can be almost anywhere between straight up and straight back, so long as it does not fall below the rostrum of the sphenoid and bypass the functional part of the nose altogether. The size and shape of this teapot,

which is the vestibule, has obsolutely nothing to do with it. It could be replaced by a little round pipe or big square tank, or a slide trombone. So long as the hole is pointed in the right direction, it does not make a bit of difference what comes before. To point the airstream below the rostrum of the sphenoid, a major distortion would be required: a flattened nose, a saddle nose, or something else that is pretty easily diagnosed. If the jet veers to one side enough to create a dry spot, and the dry spot can be shown to make trouble it should be straightened. I believe it is as simple as that.

SEVENTH INTERNATIONAL CONGRESS OF BRONCHOESOPHAGOLOGY.

The Seventh International Congress of Bronchoesophagology will be held at Kyoto (Japan) University, September 12-14, 1958, under the direction of Prof. Mituharu Goto, M.D., Otorhinolaryngological Clinic, Kyoto University Hospital, Kyoto, Japan.

A registration fee of $25.00 U.S.A. will be charged all physicians and a fee of $10.00 for wives or non-medical guests. Registered members are admitted free to banquet, receptions and sightseeing tours.

SYMPOSIUM.

MODERN CRITERIA FOR DIAGNOSIS AND THERAPY OF MALIGNANT DISEASE IN THE UPPER RESPIRATORY TRACT.

MODERATOR:

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CANCER OF THE NASOPHARYNX AND OROPHARYNX.

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An enlarged lymph node in the cervical area is frequently a metastasis from a quiet primary carcinoma. The diagnosis is properly made by identifying the primary lesion, not by removing or sampling the node. If thorough visual and tactile search reveals no primary, multiple biopsies from the innocent appearing nasopharynx may reveal it. In addition a submental-occipital view of the skull base should be obtained. An infiltration arising from the lateral nasopharynx can produce distinctive alterations in this view, though nothing is seen, felt or obtained by biopsy in the nasopharynx.[1] In any case, this sequence properly precedes direct approach to the nodal enlargement.

When the latter is necessary the use of the needle biopsy

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offers many advantages over node excision. A new needle has recently been described for this purpose.[2]

CANCER OF THE NASOPHARYNX.

Cancer of the nasopharynx constitutes 0.4 per cent of all cancer in occidental countries;[3] it constitutes 5 per cent of cancer in China.[4] The reason for the difference is not known. In order of frequency the symptoms of cancer of the nasopharynx are: *1.* Enlarged gland or glands in the neck; *2.* nasal symptoms, obstruction or epistaxis; *3.* Vth nerve pain (in clinical analysis of this symptom a routine submental-occipital X-ray view is a useful addition to clinical examination); *4.* signs of auditory tubal obstruction; and, *5.* paralysis of cranial nerves other than the Vth cranial nerve.

In 50 per cent of cancers of the nasopharynx a metastatic node is the first evidence of disease. In 70 to 80 per cent metastatic nodes are present at the time of diagnosis. A high posterior cervical triangle node is the most frequent individual nodal involvement.[5]

The average patient waits three months after the appearance of first symptoms of cancer of the nasopharynx before consulting a doctor; the correct diagnosis is not made for an additional four months in the average case; and proper therapy is not applied to the average cancer of the nasopharynx until ten months have elapsed from initial symptoms.[6] While 10 to 15 per cent of cases present cranial nerve paralyses as the first symptom of disease, nerve involvement has increased to 20 to 25 per cent of all cases by the time correct treatment is initiated.[3]

Lack of microscopic differentiation of a high percentage of nasopharyngeal cancers has resulted in varied pathologic classifications. Division into squamous cell epitheliomas, lymphoepitheliomas, and lymphosarcomas delineates treatment and prognosis satisfactorily. The above order is that of frequency of occurrence.[1] Favorable prognosis, however, is in reverse order. Thirty to 50 per cent of lymphosarcomata can be expected to be well five years after treatment. This is

reduced to 25 per cent with lymphoepitheliomas and to 15 per cent with squamous cell carcinoma.[1,5,7]

Cranial nerve involvement, decalcification of the skull base, and presence of metastases are ominous prognostic signs in a given case.[7]

Cancer of the nasopharynx cannot be cured by surgical methods. Principal dependence is on radiation therapy. There is a wide variance in methods of applying it.

Since diagnosis centers on the surgical techniques of the otolaryngologist he frequently maintains an interest at the therapy level. This is usually expressed in terms of surgical exposure of the tumor, as with palatal fenestration[8] or partial septectomy;[9] in fulguration of the tumor; or in techniques of intracavitary radiant applicators.[10,11] The primary tumor that persists to kill the patient, however, is that cell most remote from the cavity and from fulguration and local radiant sources. For more complete blanketing and homogeneous distribution through the tumor zone it is probably true that external, multiple-port irradiation most nearly fulfills the ideal.[7,12,13] Combinations of intracavitary and external ray sources have no advantage and some disadvantage to deliverance of the total dose by multiple external ports as the primary treatment. The intracavitary techniques are most applicable for tumor persistence where the skin will tolerate no further external irradiation.[9]

Because of skeletal parts that must be traversed by the beam, the increased penetration of the Gamma rays of cobalt 60 are preferable for external treatment to the radiations of the more universally available 250 kilovolt equipment.

Since 70 to 80 per cent of cancers of the nasopharynx have or develop cervical nodal metastases treatment of the anterior and posterior cervical triangles should be included with treatment of the primary. Because over 30 per cent of cases metastasize eventually to the contralateral side also, the supraomohyoid cervical lymphatics should be included on that side. If metastasis is already present on this second side the field should be extended to the clavicle.[13]

Total irradiation dosage with any technique is a factor of individual response. Here the real skill of the radiation therapist becomes apparent. The otolaryngologist serves best who is most critical in his selection of a therapist.

The usefulness of neck dissection for persistent metastatic node involvement has to be analyzed in the light of two considerations: *1.* The uncertainty of primary control; *2.* the unresectable lateral pharyngeal node, the highest node in the chain. A case in this predicament requires the application of the most thorough irradiation effort to the metastasis before surgical resort.[14,15]

Persistence of primary tumor, as evidenced by reappearance after a quiet interval, should be pursued by additional irradiation. The technique may have to be modified by skin tolerance. This may be carried to a third and a fourth series. Thus, though "cure" and "survival" are not synonymous, the latter is desirable to the limit of a reasonably comfortable life. In one small series the three year survival was increased from 21 per cent to 57 per cent by these persistent tactics.[16]

CARCINOMA OF THE OROPHARYNX.

The oropharynx is that anatomic segment extending from the arch of the atlas to the hyoid bone. Its anterior limit is the buccopharyngeal sphincter comprised of anterior faucial pillars and soft palate, and the base of the tongue to which the sphincter closes. Malignancy in the area occurs principally as: squamous carcinoma of the soft palate; squamous carcinoma, lymphoepithelioma, and lymphosarcoma of the tonsil; squamous carcinoma, lymphoepithelioma, and lymphosarcoma of the lingual base; and squamous carcinoma of the linguo-tonsillar and glosso-pharyngeal sulcus and vallecula. Malignancy of the posterior oropharyngeal wall and of the lateral wall posterior to the posterior faucial pillar is uncommon.

Cancer of the Soft Palate.

Carcinoma of the soft palate is typically squamous and differentiated. True primary involvement, as distinguished from extension of tonsillar cancer is not common. Metastasis,

when it occurs, is to the upper deep cervical nodes. External irradiation therapy may be combined with irradiation through the open mouth. Neck dissection should be done for uncontrolled metastases if the primary appears controlled. The five-year control rate through these treatment methods approximates 25 per cent.

Cancer of the Tonsil.

Cancer of the tonsil comprises 2 to 3 per cent of all cancer. Only cancer of the laryngopharynx is more common in the upper respiratory tract. Lymphosarcoma occurs on an average at an earlier age than does lymphepithelioma or squamous carcinoma. The latter occurs preponderantly (nine to one) in men. It usually spreads quite rapidly to the soft palate, the anterior pillar, or the linguo-tonsillar sulcus. Metastasis to the upper deep cervical lymph node is usually present. It may be the outstanding first symptom with an inconspicuous primary. The latter may be palpable in the tonsil when it is not actually visible. A punch biopsy of the suspected area is a sounder approach to diagnosis than is enucleation of the tonsil. The lymphatic flow from the malignant area will increase in direct proportion to the trauma and extent of the biopsy procedure.

As a method of primary treatment surgery has no place in the treatment of tonsillar cancer. External irradiation is the best therapy. Bilateral fields include the immediate upper deep cervical lymphatic area. The best assumption is that these are involved metastatically even in the absence of clinical evidence. The metastatic disease is more uniformly responsive in tonsillar cancer than in other oropharyngeal cancers. This is the most important part of the disease, and treatment should be carried to full dosage even though the nodes disappear early in the course of treatment. Neck dissection is indicated for the infrequent case where there is primary control with persistent metastases. The dissection must be carried well above the level of the digastric muscle.

One of the most distressing complications of the irradiation treatment of tonsillar cancer is mandibular radionecrosis. It may develop years later. The usual immediate cause is ex-

tension of infection from carious teeth and peridental infection into the partially devitalized bone. This can be avoided by the removal of the teeth before therapy is begun.

The presence of metastases in cancer of the tonsil has an important bearing on prognosis. In one series of 160 patients metastases were present in three-fourths of the cases. In 11 per cent these were bilateral. The five-year control rate in the absence of metastases was 20 per cent. In those cases presenting unilateral metastases it was 4 per cent. When bilateral metastases were initially present the salvage at the end of five years was zero.[17]

The best prognosis in tonsillar cancer is in the lymphosarcomata. The outlook is approximately twice as favorable as with squamous cancer, 30 to 40 per cent five year control as compared with 15 to 20 per cent. The lymphoepitheliomata lie somewhere between.

Cancer of the Lingual Base.

That portion of the tongue posterior to the circumvallate papillae has well known histologic differences from the mobile anterior lingual two-thirds. Cancer involves the posterior area less frequently, in a ratio of five cancers in the anterior segment to one posteriorly. Cancer in the lingual base is a disease primarily of males. Squamous cancers far outnumber the lymphoepitheliomata and lymphosarcomata. Upper deep cervical node metastases (at and below the level of the hyoid horn) are the rule in all three types, 80 to 90 per cent have metastases. The spread is often bilateral, in one-third if the primary is confined to one side of the base, and in two-thirds when the primary is on both sides of the midline.

External irradiation is easily applicable to the primary, and necessarily crosses both lateral lymphatic zones. Special applications of radium needles and radon seeds have been advocated,[18] but on theoretical grounds lack homogeneity and add nothing in total dose to what is accomplished through external irradiation. Unfortunately, in the squamous cancers five-year control of the disease is obtainable in fewer than 10 per cent of cases by either method. Again, the results are twice as good with lymphoepithelioma and lymphosarcoma.

Surgical resection of the primary area is not feasible in cancer of the lingual base. Since X-ray sterilization of the primary occurs only infrequently, there are few instances where neck dissection for the uncontrolled metastatic bed is indicated in this miserable disease.

Cancer of the Linguo-Tonsillar and Glossopharyngeal Sulcus and Vallecula.

Occurring, as they do, in a natural anatomic fold these tumors can be overlooked with the flashlight-tongue blade routine of the casual examiner. It will be a real accomplishment when the otolaryngologists indoctrinate their medical conferees in the simple routine of mirror examination and forefinger palpation of the oropharyngeal area. Sulcus cancer can be deeply infiltrating and present only a fissure-like ulcer at the surface.[19,20]

As with all of the oropharyngeal cancers, the sheltering effect of the mandible and the proximity of the cartilaginous larynx demand careful external irradiation of sulcus and vallecular cancer. Penetrating Gamma irradiation, as with Cobalt 60 on theoretical grounds, offers distinct advantages.[21,22,23] Tumor residuum is treated by some with implantation of radon emanation seeds. Special techniques with radium needles and emanation seeds have their greatest usefulness where further external irradiation for persistent tumor is not possible because skin tolerance would be exceeded.

Surgery is useful for persistent metastasis if the primary is controlled. This combination of circumstances is not common. In addition the metastatic involvement has a way of infiltrating the carotid sheath. In the presence of uncertain primary control, the prospect of carotid resection *en bloc* is discouraging to temperate surgical minds; indeed, it is a rare cancer in this area that lends itself to surgery for the primary or the secondary. Sulcus tumors do not have as favorable outlook for cure as does tonsillar cancer. As a group, they do, however, have a better outlook than cancer of the lingual base.[7]

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THE NASAL CAVITY AND PARANASAL SINUSES.*†

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A discussion of cancer of the nasal cavity and paranasal sinuses which does not begin with an admission of the distressing fact that the results of treatment are generally poor, is simply not realistic. One reason for the poor results is anatomic; the structural continuity of the area is an invitation to rapid spread of any disease process. Another reason is clinical; the usual textbook description, though unfortunately it very often fits the state of the patient when he is first seen, is the picture of advanced disease, not of early, curable disease. Diagnosis will be delayed and treatment will be ineffective until this mistaken concept is overturned.

Patients are generally late in seeking medical advice. A delay of three months is about average, but a delay of six to nine months is not at all unusual, and every series contains cases in which the delay is a year or more. One reason why patients delay is that the symptoms of cancer in these areas are often the symptoms of diseases which they have had for months or years. The etiology of malignant disease of the nasal cavity and paranasal sinuses is still unsettled, but in many instances it is related to such conditions as chronic sinusitis, allergies of various kinds, polypi, and nasal obstructions in which there is interference with proper ventilation and drainage, and to which the patients have become accustomed.

Another reason why patients are seen late is that the physicians and dentists, whom they have first consulted, have treated them on the basis of the symptoms they presented, without investigating what underlies them. Otolaryngologists themselves are often at fault. We plead for the early refer-

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ence of patients with malignant disease, but when our desire is accomplished, we often lack the index of suspicion which would direct us to the investigation which in turn would lead to the diagnosis.

The early symptoms and signs are in no way indicative of the lethal disease which is causing them. Unilateral epistaxis, nasal obstructions and discharges, and widening of the nasal bridge are by no means uncommon in nonmalignant disease. Even when the symptoms and signs become more severe; pain radiating to the teeth, the cheek and the orbit, ocular proptosis, and tender swellings of the palate and antral area; the physician who does not possess a healthy curiosity will not undertake the appropriate diagnostic studies.

Malignant disease of the nasal cavity and paranasal sinuses must be constantly suspected as an explanation of such symptoms and signs as I have just listed. It must be considered to be present until it is proved not to be, under the following conditions:

1. When unilateral masses are present in the nose.

2. When polypoid masses have been present in the nose and sinuses for a considerable time, and their removal is attended with abnormally free bleeding.

3. When there is prolonged unilateral nasal blockage, with a blood-tinged purulent discharge.

4. When the complaint of pain in the cheek or upper teeth is disproportionate to the clinical findings.

5. When a purulent sinus infection fails to respond to adequate treatment within a reasonable period of time.

6. When unilateral swelling and tenderness occur in the nasal area.

These symptoms and findings require a diagnostic survey, in which a careful local examination and nasopharyngoscopy are the first steps.

Roentgenologic examination comes next. The positive signs include opacity of the nasal cavity, irregularity of the soft

tissues, and bony destruction. Cytologic examination by the Papanicolaou technique is valuable when it is positive, but useless and possibly misleading when it is negative. The same holds for aspiration biopsy.

Even frozen section has a margin of error, yet there is no room for error when mutilating surgery, such as is necessary in this variety of cancer, must be undertaken. If repeated biopsies are negative and the suspicion of cancer still persists, an exploratory procedure is warranted. The pathologist must be supplied with a representative section of tissue, and he must be given ample time to study it.

These facts may be elementary, but they constitute the background of successful therapy. Until the disease is recognized, treatment will not be instituted. Unless the disease is recognized early, the most potentially useful treatment will be useless.

Irradiation, which was formerly generally employed, was used, as Frazell has well put it, more by default than by any proved superiority of results; in fact, it has decided disadvantages, including severe ocular complications. It may also cause infection and prolonged disability because of the edematous obstruction of normal drainage channels by infected and necrotic cancer tissue. The corrective procedures required when this happens are often of such magnitude that radical surgery might as well have been employed to start with.

It is true that with the advent of supervoltage therapy, irradiation promises for the future better results with fewer disastrous side-effects. That day, however, has not yet arrived. At the present time, the optimum treatment is radical surgery combined with maximum doses of radiation by both the intracavitary and external techniques.

The surgery must be radical. Anything less than wide exposure and *en bloc* removal of the involved tissues is inadequate treatment. Surgery of such magnitude is now practical because of improved techniques of anesthesia, including hypotension, the liberal use of whole blood provided

by hospital blood banks, and the availability of wide-spectrum antibiotics.

The Weber-Fergusson incision is used to expose the facial surface of the maxilla, and the entire maxillary bone, with the maxillary sinus, ethmoids, and orbital contents, is removed *en bloc*. To accomplish this, the palate must be split in the midline and the maxilla and portions of the zygoma and ethmoid bones separated from their attachments. The sphenoidal, frontal, and posterior ethmoids are curetted and fulgurated, as are any other suspicious areas. The cribriform plate may be removed and the underlying dura cauterized, to care for possible extensions. The eye is always included in the dissection unless an obviously clear margin of normal tissue can be demonstrated between it and the lesion. If there is any doubt, the eye must be sacrificed.

Since cancer of the nose and sinuses is characteristically late in metastasizing, radical neck dissection, although it can be performed with maxillectomy, is usually deferred. Often it is not employed until the indication for it becomes evident.

During the operation, the entire cavity is irrigated with a cancerocidal solution, to reduce the number of viable cancer cells in the wound washings. When the tumor is highly malignant, it is well to follow its surgical removal with some appropriate chemotherapeutic agent. Perfusion techniques, which have given very promising results in some regions of the body, for anatomic and other reasons do not seem applicable at this time to cancer of the nasal cavity and paranasal sinuses.

The solution to this particular problem lies with the general practitioner, the dentist, and sometimes the ophthalmologist even more than with the otolaryngologist. It is our duty to be eternally on guard, ourselves, and to acquaint those who are not practicing this specialty with the real facts of this disease; otherwise, many a patient will continue to lose his only chance of life. Until diagnosis is achieved earlier, radical surgery followed by maximal radiation, which, given a fair chance, *can* control the disease, will often prove palliative rather than curative, and the results will be poor as well as

mutilating. With earlier diagnosis and correctly applied therapy, the proportion of five-year results will readily be doubled.

CANCER OF THE LARYNX AND PHARYNX. DIAGNOSIS AND TREATMENT.*

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I. Introduction.

My part of the symposium is to discuss the diagnosis and treatment of squamous cell carcinoma of the larynx and hypopharynx. I can only outline our experiences with this disease in the time allocated.

II. Classification.

We classify cancer of the larynx and the surrounding anatomical areas according to the International Radiological Congress' proposals made in Copenhagen in 1953. Of the many proposals for classifying cancerous lesions of the larynx and hypopharynx, the above appears to be workable and useful. Frequently, clinicians will make modifications in any classification according to their needs and to the needs of their patients.

Cordal, supraglottic, subglottic, hypopharyngeal and postcricoid are descriptive terms used for anatomical areas in which lesions are placed. Often the dividing line between these areas is not sharply defined, and in some patients it is difficult to determine the true anatomical site of the origin of the lesion. Lesions are further graded in Stages I to IV. Such staging is an aid to the clinician in planning therapy and evaluating results. The staging of lesions by the clinician is an attempt to determine whether impairment of function

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exists in any part of the larynx or hypopharynx and whether there is direct extension or metastases. Other characteristics such as size, presence of edema, surface growth, or deep ulceration may be of further assistance in classification.

III. Diagnosis.

The diagnosis of cancer in the areas under discussion can usually be made by biopsy and microscopic examination of the tissue. Other aids in establishing diagnosis are, Papanicolaou's stains of extruded cells, tomography, contrast media Roentgen studies and palpation. Further surgical procedures may be required for correct diagnosis such as repeated biopsies and thyrotomy. If it is necessary to resort to the latter, the operating room should be set up and the surgeon prepared to carry out definitive surgical treatment after the diagnosis is established by microscopic frozen sections.

In our experience, over 99 per cent of the cancerous lesions in this area have been of the squamous cell variety. Generally, if the site of origin of the lesion is other than the true vocal cords, one may expect to find the neoplasm less well differentiated microscopically. According to Broder's classification, most cordal squamous cell carcinomas are of Grades I and II. Only recently, we have seen a case with a small cancer of the true vocal cord which was remarkably anaplastic in its growth patterns, and was considered by our pathologist to be Grade IV.

IV. Treatment.

Surgery and radiation therapy are the only two forms of therapy available today for the treatment of patients with cancer of the larynx. In no sense of the word, should these two methods of treatment be competitive, but should be closely integrated even in the same patient when indicated.

Illustrative cases with various types of lesions will be presented.

Patients with early Stage I cordal squamous cell carcinomas should be treated by peroral removal and fulguration, rather than with radiation therapy.

Cordal lesions, Stage I, slightly more advanced than in the above cases, are ideal for either radiation therapy or laryngofissure. I cannot specifically recommend surgery or radiation therapy for a given patient since many factors, such as age of patient, general health, etc., will influence the doctor's choice of treatment. Patients with carcinomas *in situ* localized to a small area of the true vocal cord should be treated surgically.

If we consider more extensive lesions of the vocal cord, such as a cordal lesion, Stage II, where function is impaired and there are no palpable cervical lymph nodes, surgery should be considered in lieu of radiation therapy. The Som[1] operation for posterior one-third cordal lesions, and hemilaryngectomy or the modification of this procedure with primary skin graft, as advocated by Figi[2] should be considered.

In patients who have a cordal lesion, Stage II, (even with both vocal cords involved), with marked limitation of motion and the cancer is still confined within the cartilaginous larynx, the procedure of choice is widefield laryngectomy. Patients with late Stage II and III cordal lesions will require widefield laryngectomy and simultaneous unilateral block dissection. Many of these cases with advanced lesions will have ventricular or subglottic extension or both. Cancer arising on both vocal cords may be so extensive that a later contralateral block dissection may be necessary if metastatic nodes appear.

Patients with supraglottic squamous cell carcinoma are usually first seen in late Stage II or Stage III; however, occasionally a patient with an early Stage II lesion in this area will be seen. In our series of cases, we have seen none with a Stage I lesion in these areas. Stage II ventricular lesions require widefield laryngectomy. Stage II false cord lesions, particularly if their growth is exophytic, will require widefield laryngectomy. Stage II false cord lesions have potentially metastasized to the cervical lymph nodes when the patient is first examined. Such individuals must undergo widefield laryngectomy and elective block dissection. Further, metastases may occur on the contralateral side of the neck, and patients must be examined carefully and frequently following

the initial surgery, so that contralateral block dissection may be carried out as early as possible if nodes appear.

Advanced Stage II supraglottic lesions in the region of the petiolus of the epiglottis are difficult to treat by any method. These lesions must be considered as potentially metastasizing to the lymph nodes in both sides of the neck. Widefield laryngectomy plus initial unilateral block dissection of the neck, and later block dissection of the contralateral side are frequently indicated. This is particularly true when microscopic lymph nodes are found in the original operative specimen. Patients with Stage II lesions on the lateral surface of the epiglottis and upper aryepiglottic fold areas, must be considered candidates for similar surgical treatment. Cases with Stage II lesions of the aryepiglottic fold proper extending into the false cord and pyriform sinus must be considered for similar treatment. This is true also for patients with Stage II lesions in the arytenoid area. All patients with Stage III lesions in those areas should be treated likewise, providing the lymph node metastases are resectable.

Patients with lesions in other portions of the epiglottis, not noted above, should be treated surgically. Cases of cancer of the free upper surface of the epiglottis suitable for peroral removal are rare indeed. Such lesions would fall into Stage I, and we have seen none in our series of cases. Likewise, patients with lesions in this area, early Stage II, suitable for transhyoid resection are rare. We have seen one patient who had an extensive Stage II cancer in this area which was treated by radiation therapy in 1950. In 1956 there was a recurrence on the free upper border of the epiglottis which was removed surgically by the transhyoid approach.

Patients with lesions of the entire epiglottis are usually seen in Stages II and III. Their prognosis is poor, even with extensive surgery or radiation therapy, or with both. Patients with early Stage II lesions of the central posterior surface of the epiglottis should be treated by widefield laryngectomy. Unilateral lesions of the epiglottis are most often seen in late Stages II or III, and require widefield laryngectomy in combination with unilateral or bilateral block dissection.

Pharyngeal and postcricoid lesions will be considered next. Surgery is the treatment of choice for these. A high percentage of patients with these lesions fall into Stage III when first examined. Hence, most all of the cases with pyriform sinus lesions require some form of extensive pharyngeal and laryngeal surgery and block dissection of the neck. Patients with early Stage II lesions of the lateral hypopharyngeal wall carry a good surgical prognosis, providing partial pharyngectomy and block dissection are carried out in continuity. Patients with posterior hypopharyngeal lesions encroaching upon the upper esophagus require extensive surgery, both as to the local primary lesion and to the lymph bearing areas on both sides of the neck. In addition, plastic repair of the pharyngeal and esophageal defects must be done. The extent of such lesions usually precludes the saving of the larynx itself. Since a high percentage of these cases have palpable cervical lymph nodes on initial examination, it must be decided by the surgeon whether the nodes are resectable before surgery is attempted.

In our series of cases, we have had only two patients with primary subglottic squamous cell carcinoma. One patient's lesion was classified late Stage II while the other's was classified as Stage III. In neither patient was the disease arrested, in spite of extensive surgery. Subglottic lesions metastasize early to the lymph channels in both sides of the neck and upper mediastinum. As far as our limited experience is concerned, we believe that any known form of treatment for Stage II or Stage III subglottic squamous cell carcinoma is inadequate. This is in contrast to those cordal cancers which have extended directly to the subglottic areas.

V. Conclusions.

Many patients with carcinomas of the larynx and hypopharynx are still being examined initially in later stages of the disease, when only palliative therapy could be recommended. In fact, approximately 20 per cent of our cases were classified as late Stage III and Stage IV. The remaining 80 per cent of the cases underwent treatment directed at arresting the disease. Surgical treatment was the treatment of choice in most instances.

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MODERN CRITERIA FOR DIAGNOSIS AND THERAPY OF MALIGNANT DISEASE IN THE UPPER RESPIRATORY TRACT.*

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Melanoma of the head and neck presents us with significant clinical diagnostic, therapeutic and statistical data that warrant careful analysis. Approximately 25 per cent of all the melanomas of the body occur in the head and neck area. This of course, highlights the importance of diagnosing this condition accurately. Fifty per cent of all the patients that we see, with melanomas in the head and neck give a history of having had a brown or black mole present all of their lives. This means that you cannot disregard the lesion which has been present for a 20-, 30-, 40-year period. Fifty per cent of the melanomas that we see give a history of repetitive trauma; not a single incidence of trauma, but repeated trauma, whether shaving, cleaning the teeth, combing the hair, at the collar line on the neck, in the mouth, on the lips, etc. The same situation applies to fingers, feet, penis, anus, from the brassiere strap on the shoulder, from the belt buckle at the waist, etc.

As far as the diagnosis is concerned, we feel that any change in color or clinical appearance, or any subjective change such as itching, stinging or burning, warrants careful observation of the pignmented lesion. Certainly if the pigmented lesion begins to grow, if it gets blacker and larger, and if there is

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ulceration, it has to be considered as a melanoma. Those which do not have such dynamic change in their biology of course are suspicious, but those that change in color and size, and which ulcerate, have to be considered as melanomas. Here I think we must be careful not to be trapped by a condition which resembles this, called the pigmented basal cell cancer; also we have to be careful not to be trapped by the amelanotic melanoma which resembles the ulcerating squamous cell cancer but grows so rapidly without pigment mobilization that it may be confused with the squamous cell cancer.

I think the other point in the differential diagnosis is what to do about the innocent or benign pigmented lesion, the neuronevus. The most important factor here is separating the nevi which can undergo malignant deterioration from the ones which ordinarily do not. This is an important clinical observation. The nevi that are puckered like a wart, the nevi that have large hairy deposits in them—two or three big black hairs coming out of them—hardly ever undergo malignant deterioration, and I don't think that it is necessary to remove these prophylactically in anyone, unless it is desired for cosmetic purposes; however, the nevi that are flat, or smooth, either black, or dark brown, or blue-black, or buff colored, ordinarily can be troublemakers. No one can tell with the naked eye the differentiation between an epidermal nevus, a dermal nevus, a subdermal nevus or a junctional nevus. Actually, I think the feeling at the present time is that melanoma comes only from the junctional nevus, but those that we are interested in are the flat ones, not those that are puckered up like a wart and contain hair.

As far as the biopsy is concerned, no incisional technique should be carried out. In other words, you should not cut into this black ulcerating area to obtain tissue for biopsy. This is a very strict rule. If the lesion is ulcerating, you may apply fibrin foam or wash it and have a Papanicolaou examination done. I believe the safest thing, however, is to do an excisional biopsy of the entire area. In other words, you are at least a centimeter or two around the peripheral margin of the lesion you suspect, and this is sent *in toto,* en masse, to the laboratory. If it is reported as a benign lesion, that is the end of

the procedure. If it is reported as a malignant melanoma then I think you are obligated to go back and do a wider local resection, because no excisional biopsy technique is considered adequate treatment for malignant tumor.

So far as the treatment is concerned, irradiation is not effective. The salvage rate here approaches zero. Surgical excision is certainly the method of choice in all cases, when possible. The head and neck, fortunately, lend themselves better to technical excision than almost any other area of the body. We are not faced with the complex problem here of an individual with a melanoma on the sole of his foot or on his big toe, which poses the question of whether the entire leg should be amputated or disarticulated. Our areas are neat and tidy as far as regional activity is concerned. My own personal feeling is that if the melanoma is less than 2 cm. in size and there is no evidence of clinical metastasis, and if it is not necessary to operate in the neck to excise it, I no longer feel that any continuity resection is indicated. I do a wide resection of the primary lesion. If the melanoma is large—over 2 cm. in size—regardless of its position, but particularly if it is located on the neck, then an elective neck dissection is included in the absence of obvious metastatic disease.

If there is evidence of metastasis in the neck, the neck dissection is always included with the excision of the primary lesion. The salvage rate in our group of cases is not significantly higher where elective neck dissections have been done in the treatment of what we classify as situations with a high possibility of microscopic metastatic melanoma. I think the difference is only about 5 per cent and that is the reason I have retreated to a more reasonable position.

Unfortunately, melanoma can metastasize by any of three routes. Its most frequent mode of metastasis is hematogenous. When it does metastasize by the hematogenous route, the prognosis is hopeless. The next most common is in the regional lymphatic bed. Here the prognosis is between 25 and 30 per cent for the five year cure. I think that if we will appreciate that originally this disease was considered to be almost totally hopeless from a prognostic point of view, a 25 per cent cure is a reasonable figure; however, I would like to

state that five years in melanoma is not the answer to the problem because at the end of ten years the 25 to 30 per cent figure has dropped down to about 15. The third method of spread is in the subdermal lymphatics, and when melanoma behaves like this, it is, in my experience, practically uncontrollable.

I would make one statement about melanoma in children. As you know, the incidence of malignant tumors in children is very low. Shortly after birth it is about 20 per 100,000. Cancer in children is extremely rare. Melanoma does occur in children, but it does not have the same clinical significance as it has in adults. It does not metastasize as readily. It is usually handled by a local extirpative operation. Consequently, most of the nevi we see in children can be handled with impunity.

Melanoma of the oral cavity or any mucous membrane area has had almost a uniformly fatal ending.

ANNUAL OTOLARYNGOLOGIC ASSEMBLY UNIVERSITY OF ILLINOIS.

The University of Illinois College of Medicine Department of Otolaryngology announces its Annual Otolaryngologic Assembly from September 29 through October 5, 1958. The Assembly will consist of an intensive series of lectures and panels concerning advancements in otolaryngology, and evening sessions devoted to surgical anatomy of the head and neck and histopathology of the ear, nose and throat. Interested physicians should write direct to the Department of Otolaryngology, 1853 West Polk Street, Chicago 12, Ill.

THE NOSE AND PARANASAL SINUSES.*

HEINRICH KOBRAK, M.D.,
Detroit, Mich.,

and

G. EDWARD TREMBLE, M.D.,
Montreal, Canada.

About a year and a half ago, Dr. Heinrich Kobrak approached me to see whether I would collaborate with him on a film. He had in mind a movie of the nose and sinuses. It was decided to show some of the anatomy and physiology of the nose and to conclude with a few clinical cases. On his suggestion I was to supply the animation, pictures of ciliary movement and some of the cases. Dr. Kobrak was to take pictures of the skull to show the sinus openings and also some common conditions encountered. It was thought that the film would be of use to senior students and internes. A number of meetings were held to place the sequences in their proper order. The last time Dr. Kobrak was in Montreal was about the middle of September. He was confident the film with the narration would be ready for the Academy meeting in Chicago in October. Two days before the Chicago meeting, I received a letter from him asking me to meet him to go over the picture before it was shown at the general meeting.

On arriving in Chicago, I learned the sad news that Dr. Kobrak had suffered a fatal coronary attack a day or so before the meeting. After searching his home and laboratory without avail, the film was finally found in the back of his car. Whether he had just received it after being processed or whether he intended to drive to Chicago, I do not know. As a result, it was up to me to carry the ball alone, so to speak.

When I saw a preview of the film I was a bit disturbed. Parts of it were overexposed, especially the microscopic slides.

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*Read at the meeting of the Eastern Section of the American Laryngological, Rhinological and Otological Society, Philadelphia, Pa., January 9, 1958.

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I had suggested some lateral views of the normal nose, showing the sinus openings and the opening of the naso-lacrimal duct. In other words, to my mind the picture could be improved and parts of it needed re-editing. I have some film showing air currents through the nose and ciliary action in the presence of an acute cold, which I think would enhance its value; however, I just received the movie from New York about ten days ago, and I have not had time to add these sections. I am only sorry that Dr. Kobrak is not here when it is being shown in its original form.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The next joint annual meeting of the North Carolina Eye, Ear, Nose, and Throat Society and the South Carolina Society of Ophthalmology and Otolaryngology will be held in Asheville, N. C., on September 14-17, 1958, at Grove Park Inn. An excellent program is in the making. The following guest ophthalmologists will be present: Dr. James A. C. Wadsworth, New York, N. Y.; Dr. Arthur Gerard DeVoe, New York, N. Y.; and Dr. Frank B. Costenbader, Washington, D. C. The following guest otolaryngologists will be present: Dr. F. W. Davidson, Danville, Pa., and Dr. Tom Rambo, New York, N. Y. A third otolaryngologist will be announced in the near future. A good attendance is anticipated.

For further information write directly to Dr. George Noel, c/o Cabarrus Bank Bldg., Kannapolis, N. C., or Dr. Roderick Macdonald, 330 East Main St., Rock Hill, S. C.

IRRIGATION OF THE MAXILLARY SINUS THROUGH THE MIDDLE MEATUS.*†

MERVIN C. MYERSON, M.D.,

Beverly Hills, Calif.

This paper is written primarily to stimulate interest in irrigation of the maxillary sinus through the middle meatus, preferably by way of its natural orifice. There probably always will be some controversy regarding the relative advantage of irrigating this sinus by way of the middle or inferior meatus. It will remain, however, for the individual rhinologist, by virtue of training, understanding, and experience, to decide the technique he wishes to follow in irrigating the maxillary sinus. A few visits to the anatomy laboratory will convince the most skeptical that irrigation through the middle meatus can be performed in a high percentage of cases. Although the more generally accepted method of irrigating the maxillary sinus is by way of the inferior meatus, using a needle or trocar, the number of otolaryngologists who are using the natural orifice and middle meatus is increasing. Many rarely resort to puncture through the inferior meatus. It has also been noted that it is easier to enter the maxillary sinus by way of the middle meatus in children than in adults.

In order to appreciate the middle meatus approach one should have an understanding of the structures involved, and their relationships. At about the tenth week of fetal life the mucous membrane of the primitive middle meatus of the nose begins to pouch laterally. This evagination indicates the beginning of the maxillary sinus and is the location of the future maxillary ostium. The maxillary ostium is situated in the posterior portion of the ethmoidal infundibulum where the posterior part of the bulla tapers away on the upper side, and the dorsal end of the uncinate process does the same on the

*Read at the meeting of the Western Section, American Laryngological, Rhinological and Otological Society, Beverly Hills, Calif., Jan. 19, 1958.

†The illustrations are from the Archives of Otolaryngology, 15:80-91, Jan., 1932, and 15:716-725, May, 1932.

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lower side. All of these structures, which vary in their size, contour and relationship, lie under cover of the middle turbinate. The membranous portion of the middle meatus lies between the dorsal extremity of the uncinate process and the perpendicular plate of the palate bone, and between the dorsal extremity of the uncinate process and the ethmoidal bulla. This has been called the undefended C-shaped region by

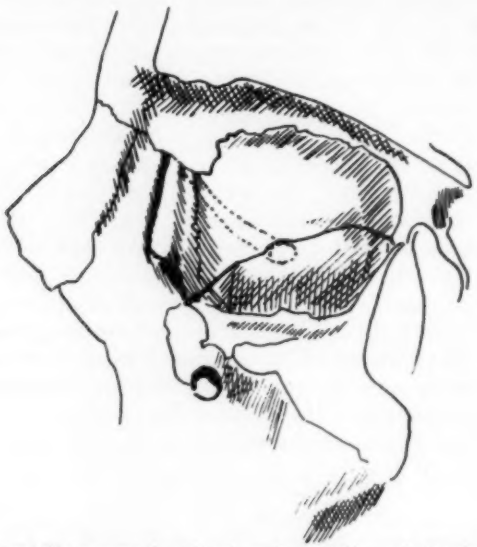

Fig. 1. Semi-diagrammatic, showing the relation of the infundibulum and maxillary ostium to the lamina papyracea, orbital plate and surrounding structures.

Schaeffer. It is in this area that the accessory ostia are found.

For success in the manipulation and introduction of a canula into the orifice of the maxillary sinus, it is important to have a knowledge of the maxillary opening and its accessory ostia, in addition to their relationship to the structures mentioned above.

The orifice of the maxillary sinus is located in the posterior

portion of the infundibulum immediately below the level of the orbital floor and, therefore, just below the lowermost portion of the lamina papyracea of the ethmoid bone (see Figs. 1, 2). The orifice is usually so placed in the infundibulum beneath the middle turbinate as to be readily approached and entered in a large majority of cases. At times it is buried deeply and may be inaccessible. When the ostium is deeply placed, it is rarely hidden by the bulla; it is in-

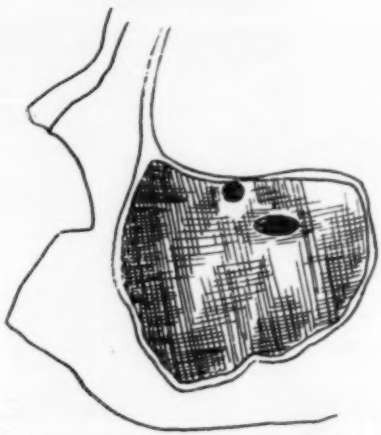

Fig. 2. Semi-diagrammatic, showing nasal wall of maxillary sinus and the usual location of the ostium; an accessory orifice is located posteriorly and below.

accessible in such cases because of the high obstructing uncinate process which governs the depth of the infundibulum. Of importance also is the distance between the uncinate and bulla, the space commonly known as the hiatus semi-lunaris. This space may be obliterated by contact of the bulla and uncinate, or it may be as wide as 3 mm. When greatly narrowed it presents an obstacle to the ready passage of a canula.

The size of the maxillary ostium varies from a diameter of 1 mm. to a measurement of 17x11 mm. Quite frequently the ostium occupies the entire infundibular region or most of it,

Fig. 3. Semi-diagrammatic, showing variety of maxillary ostia.

so that a canula can be inserted with ease. The size of the accessory orifice varies from a pinhead to 10.5x6.5 mm. (see Figs. 3, 4).

It is important to know that the position of the maxillary ostium may be vertical, horizontal or oblique. When positioned horizontally or obliquely, it indicates the beginning of a canal or passage which terminates in a vertically placed orifice in the medial wall of the maxillary sinus (see Fig. 5). These passageways have been stressed by Larroude, Simon, Van Alyea, and the author (see Fig. 6). Simon claimed that a canal exists in 90 per cent or more of maxillary sinuses.

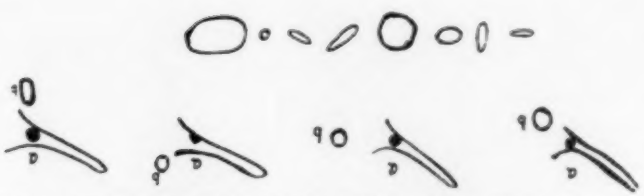

Fig. 4. Semi-diagrammatic, showing variety of sizes, shapes and relative positions of the accessory ostium.

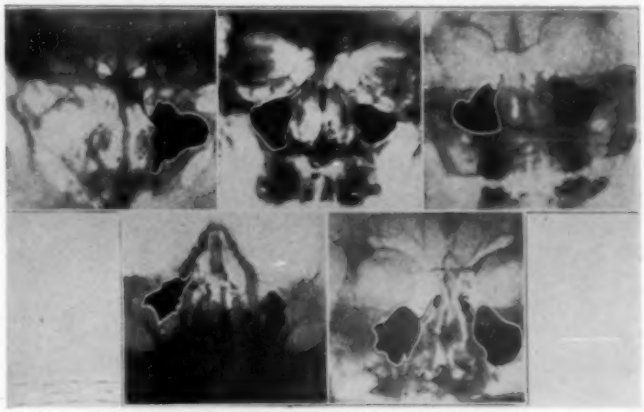

Fig. 5. Roentgenographic studies made with iodized oil, showing variety passageways into maxillary sinus.

This paper is based upon a series of 500 consecutive patients in whom an attempt was made to irrigate the maxillary sinus through its natural orifice. In those cases in which the ostium could not be entered, the membraneous portion of the middle meatus was deliberately punctured with the same blunt canula employed for entering the ostium. The natural orifice was entered in 399, or 79.8 per cent, of this series of 500 cases; in 48, or 9.6 per cent, the membraneous portion was punctured with the same blunt-ended irrigation canula. The total number irrigated through the middle meatus was 447, or 89.4 per cent, of the 500 subjects.

In order to introduce a canula through the natural opening

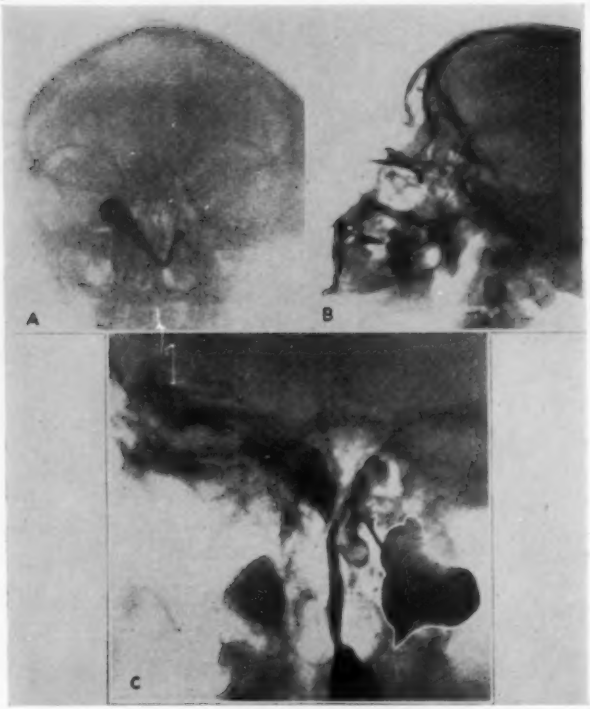

Fig. 6. Roentgenographic studies showing (A and B) canula in oblique passageway; (C) passageway after instillation of iodized oil.

of the maxillary sinus, the middle meatus and the structures traversed by the instrument must be anesthetized. To accomplish this a small flat tampon is inserted under the middle turbinate just beyond its anterior end. It is advantageous to tie a piece of cotton thread to the tampon, the distal end of which is cut off at the nostril. When anesthesia is complete, the tampon is removed by pulling on the thread. This eliminates the necessity of using an instrument in the middle meatus, which sometimes bruises the tissues because of the narrowness of the passage or the depth of the position of the tampon; also, at times, the tampon is difficult to recover.

With few exceptions, the tampon can be inserted into the region of the infundibulum; after three to five minutes it is removed and a fine cotton tipped applicator, saturated with the same cocaine solution, is placed in the same location. The cotton-wound applicator can be bent so that its distal end can be properly placed. When the middle meatus is anesthetized, an attempt is made to introduce the canula.

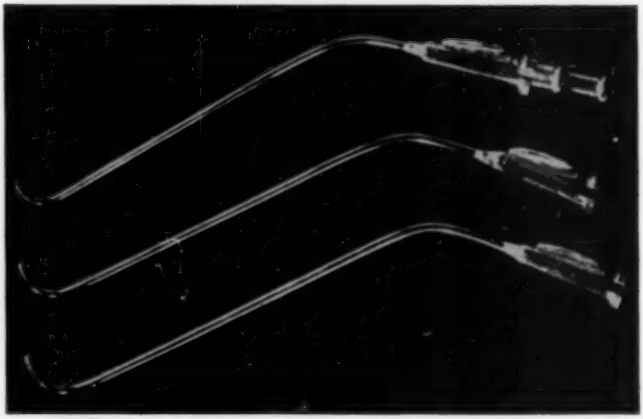

Fig. 7. Author's canulae, three sizes.

The smallest possible canula should be used when the first attempt is made to enter the ostium. The canula is introduced a few millimeters behind the anterior end of the middle turbinate with its distal end pointing upward. When the middle meatus is reached, the tip of the instrument is directed laterally; if a vertically placed ostium exists, the canula will fall readily into it (see Fig. 7). If the canula strikes the lateral wall it is withdrawn and introduced in an inverted position. In this position the distal end will fall into a horizontally or obliquely placed orifice. One can tell that the canula is in the ostium by the depth of penetration of the instrument and by the excursion of its distal end, which is controlled by the diameter of the orifice. If an ostium cannot be found the membraneous portion of the middle meatus

is perforated by the canula. Penetration through the membrane imparts a definite impulse which is readily recognized. In some cases there is a streak of blood; in most, there is none, while in others there may be no evidence of trauma. The condition of the membrane and its thickness, as well as the amount of trauma, influence the presence or absence of blood. When difficulty is encountered in the passage of the tube, one may bruise the middle meatal structures so that slight bleeding will occur for a few hours, or for as much as two or three days. The latter is exceptional and should not occur if the rhinologist is careful. When the canula is successfully introduced, there should be no injury to the tissues, nor should there be bleeding when the canula is withdrawn.

The presence of pus or mucopus in the returned irrigating fluid is evidence that the canula is in the antrum. The patient feels the fluid in his sinus, especially when the pressure of the fluid is increased. The sound heard when air is introduced into the antrum after irrigation, is distinctive.

In this series of 500 cases, the middle turbinate was impacted against the lateral wall so that the middle meatus could not be entered in 14 instances. When the space between the lateral aspect of the middle turbinate and the lateral nasal wall is so restricted, it is better to refrain from attempting to pass the canula; one should resort to puncture through the inferior meatus. At times a fine canula can be passed in an inverted position between the anterior end of the turbinate and the lateral wall, instead of beneath the anterior end of the middle turbinate, as is usually done. In five cases the middle turbinate could not be visualized because of a badly deviated, or convex septum.

It was found that patients who had nasal polyps protruding from the middle meatus presented an enlarged natural opening, so that a canula could be passed with relative ease. One is also impressed with the large number of orifices of increased diameter among the cases of chronic suppuration of the maxillary sinus.

Occlusion of the ostium during irrigation is more theoretical than real. Occlusion of the ostium occurred once in this series

because of the extremely thick exudate which was expelled with difficulty, after inferior meatal puncture. Acutely involved sinuses are never irrigated; but the exudate in cases of subacute or even chronic infections may be quite thick. This, together with the presence of a small ostium, may cause slow emptying of the sinus cavity and, at times, some pain, but the irrigating fluid will usually be returned without incident. That the ostium is rarely occluded has been proven by anatomic studies. In a study of 600 lateral nasal walls, Zuckerkandl found the smallest ostium to be 3 mm. in diameter, while the largest was 19x5 mm. In 200 sections, Oppikofer found the smallest to be 2 mm. in diameter and the largest 17x11 mm. Schaeffer and the author found the smallest to be 1 mm. in diameter.

The number of otolaryngologists interested in natural orifice irrigation has increased with the years. This is because they have found the procedure simpler and safer in a majority of cases. Schaeffer states the reason as follows: "One must bear in mind that instruments of precision, and a more highly refined technique, have come into being of late years, making successful treatment of an infected maxillary sinus by way of its natural aperture seemingly feasible in a relatively large percentage of cases." Many have stressed the advantages of irrigation through the natural orifice or through the membranous portion of the middle meatus.

Those who object to irrigation through the natural opening claim there is difficulty in locating the ostium in a large percentage of cases. They assert that the sinus cannot be properly irrigated when the return flow is partly blocked by the canula, and that instrumentation of the ostium injures and inflames its membranous lining. If the anatomy is understood and the instruments properly manipulated, these objections are not valid.

The objections to inferior meatal puncture have been known for a long time:

The procedure is frequently painful; there is usually some bleeding; there is failure to penetrate with a needle or trocar because of thickened bone.

The needle or trocar can fall into or beneath thick necrotic or polypoid membrane and cause air embolism or infiltration and emphysema of the cheek and eyelids.

Phlegmon of the soft parts of the face can occur; fluid can be injected into the pterygomaxillary fossa.

Air embolism is a well-established accident of inferior meatal puncture. Many deaths have been recorded.

An attempt was made to enter the natural opening of the maxillary sinus in 500 consecutive cases. The ostium was successfully entered in 399, or 79.8 per cent; in 48, or 9.6 per cent, the membranous portion of the middle meatus was punctured with the same canula used for entering the ostium. A total of 477, or 89.4 per cent, therefore, were successfully treated through the middle meatal approach.

Irrigation of the maxillary sinus through the middle meatus, either by way of the natural opening or by puncture of the membranous portion of the middle meatus, is a safe and simple procedure. On the basis of my experience it should be recommended as the method of choice.

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AMERICAN RHINOLOGIC SOCIETY ANNUAL MEETING.

The American Rhinologic Society will hold its fourth annual meeting in the Palmer House, Chicago, October 17-18, 1958.

Among the topics to be discussed will be pulmonary and nasal physiology, laboratory and clinical aspects of bone transplants, hump removal, roof repair, and nasal process corrections.

The preliminary program includes the following papers: "Maxillary and Premaxillary Approach to Septal Surgery," Dr. Ralph H. Riggs, Shreveport, La.; "Olfactory Factors in Experimental Neurosis in Animals," Dr. Jules H. Masserman, Chicago; "Second Golden Decade of Rhinologic Surgery—The Advances of the Past 10 Years," Dr. Harvey C. Gunderson, Toledo; "Physiology of Respiration," Dr. David Cugell, Chicago; "Concepts of Nasal Physiology as Related to Corrective Nasal Surgery," Dr. Maurice H. Cottle, Chicago; "Bone Transplants; Experimental and Clinical Aspects," Dr. Robert Ray, Chicago; and "Nasal Physiology," Dr. Irving Cramer, Cleveland. "Hump Removal, Roof Repair, Nasal Process Corrections," will be the subject of a panel; Dr. Walter Loch of Baltimore will be the moderator. The participants will be Drs. Lewis Morrison of Indianapolis, Richard Hadley of Rye, N. Y.; Charles Tucker of Hartford, and Joseph West of St. Louis.

Dr. Russell I. Williams of Cheyenne, president of the Society, will preside. Dr. Guy L. Boyden, professor of otolaryngology, University of Oregon Medical School, Portland, will be the guest of honor.

The profession is cordially invited to attend as guests. There will be no registration fee.

For further information write to Dr. Robert M. Hansen, secretary of the society, 1735 North Wheeler Avenue, Portland 17, Ore.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this information up to date).

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Erling W. Hansen, 90 So. Ninth St., Minneapolis, Minn.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Palmer House, Chicago, Ill.

AMERICAN BOARD OF OTOLARYNGOLOGY.

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Meeting: The Homestead, Hot Springs, Va., March 10-11, 1959.

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Editor, Historian, and Librarian: Dr. Edwin N. Broyles, Baltimore, Md.
Meeting: The Homestead, Hot Springs, Va., March 8-9, 1959.

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Place: The Homstead, Hot Springs, Va., March, 1959.

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Meeting: Atlantic City, June 8-12, 1959.

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Secretary: Dr. Lawrence R. Boies, University Hospitals, Minneapolis 14, Minn.
Place: The Homestead, Hot Springs, Va., 1959.

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Secretary: Dr. Robert M. Hansen, 1735 No. Wheeler Ave., Portland, Ore.
Annual Clinical Session: Illinois Masonic Hospital, Chicago, Ill., October, 1958.
Annual Meeting: October, 1958, Chicago, Ill. (Definite time and place to be announced later).

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Meeting: November 12, 1958, in conjunction with the Section on Laryngology, Rhinology and Otology of the Medical Society of the County of Kings and Academy of Medicine of Brooklyn, 1313 Bedford Ave., Brooklyn, N. Y.

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Annual Meeting: Palmer House, Chicago, Ill., October 16-17, 1958.

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Meeting:

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Meeting quarterly (March, May, October and December), on the second Thursday of the month, 6:30 P.M. at Urmey Hotel, Miami.

INTERNATIONAL BRONCHOESOPHAGOLOGICAL SOCIETY.

President: Dr. Jo Ono, Tokyo, Japan.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadephia 40. Pa., U. S. A.
Meeting: Seventh International Congress of Bronchoesophagology, Kyoto, Japan, September, 1958.

KANSAS CITY SOCIETY OF OTOLARYNGOLOGY AND OPHTHALMOLOGY.

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Meeting: Third Thursday of November, January, February and April.

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Chairman of Otolaryngology Section: Dr. Robert W. Godwin.
Secretary of Otolaryngology Section: Dr. Francis O'N. Morris.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd., Los Angeles, Calif.
Time: 6:30 P.M. last Monday of each month from September to June, inclusive—Otolaryngology Section. 6:30, first Thursday of each month from September to June, inclusive—Ophthalmology Section.

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Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Meeting: Edgewater Gulf Hotel, Edgewater Park, Miss., May 15-16, 1959.

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Meeting: Palmer House, Chicago, Ill.

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Meeting:

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President: Dr. Jose Gros, Havana, Cuba.
Executive Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa., U. S. A.
Meeting: Sixth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Time and Place: Brazil, 1958.

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Meeting: October 14, 1958, Palmer House, Chicago, Ill.

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Meeting:

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Annual Meeting:

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